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Archives of Dermatology and Syphilology

VOLUME 30 AUGUST 1934 NUMBER 2

FOLLICULAR LESIONS IN VITAMIN A AND C DEFICIENCIES

MAX SCHEER, M.D.

Assistant Professor of Dermatology and Syphilology, New York Postgraduate Medical School and Hospital, Columbia University; Adjunct Dermatologist, Mount Sinai Hospital

AND

HARRY KEIL, M.D.

Clinical Assistant, Mount Sinai Hospital and the New York Postgraduate Medical School and Hospital

NEW YORK

The recent report of Loewenthal [1] in which he described follicular papules in vitamin A deficiency provided the impetus for this communication. He recorded this lesion as a new observation because he was unable to find any reference to the condition. Medical literature has become so voluminous and complex that an occasional oversight is possible. It is in the spirit of adding to the excellent observations of Loewenthal that we have prepared this paper.

In the past year we have observed two cases of scurvy in adults in which follicular lesions were present. In one instance the resemblance of the lesions to those described in vitamin A deficiency was striking; in the other there were sufficient clinical and pathologic differences to justify a separation of the two processes.

REPORT OF CASES

CASE 1.—H. E., a spinster, aged 46, was admitted to the medical service of Dr. B. S. Oppenheimer on July 9, 1932. She had voluntarily restricted her diet for three years. She had eaten no fruit for at least six months, subsisting chiefly on chicken, dry cereals and milk. Six weeks before admission, pains in the legs and ecchymoses developed. In the week preceding entrance to the hospital she had sore and bleeding gums. Examination revealed that the patient was undernourished and emaciated. The teeth were decayed and broken, with swollen hemorrhagic areas at the gingival margins and a large hemorrhagic spot over one molar tooth. The knees and tibias were markedly tender. Over the calves and especially over the popliteal fossae there were large ecchymotic lesions averaging 2 cm. in diameter, with two or three broad confluent hemorrhagic areas. The hair follicles of the lower part of the thighs and the upper part of the legs revealed a peculiar kera-

From the dermatologic services of Drs. W. J. Highman and I. Rosen, Mount Sinai Hospital.

1. Loewenthal, L. J. A.: A New Cutaneous Manifestation in the Syndrome of Vitamin A Deficiency, Arch. Dermat. & Syph. **28**:700 (Nov.) 1933.

totic condition with many curled and broken-off hairs. There was no clinically discernible inflammatory element associated with the lesions. The diagnosis of scurvy was verified by the immediate and remarkable improvement of the patient on a diet composed chiefly of orange juice. After a stay of less than two weeks, she was sent home because of unruly behavior.

Comment.—The diagnosis of scurvy was justified by the history of dietary indiscretion, the broad areas of hemorrhage in the popliteal fossae, the adjacent ecchymoses over the calves, the spongy, bleeding gums, the painful muscles of the calves and the immediate response to the antiscorbutic diet. The follicular lesions were similar to those described in vitamin A deficiency (Frazier and Hu [2]). Although the patient had no obvious xerophthalmia, the possibility of a coincidental deficiency in vitamin A cannot be eliminated entirely, especially in a patient who had restricted her diet so carefully for three years. We were unable to make extensive observations concerning the effect of the antiscorbutic diet on the follicular eruption. It is possible that the earliest follicular lesion of scurvy may be clinically indistinguishable from that seen in vitamin A deficiency. However, the characteristic perifollicular papule of scurvy (case 2) revealed such striking differences as to warrant separation from the lesion observed in vitamin A deficiency.

Case 2.—M. S., a man, aged 42, entered the medical service of Dr. L. Kessel on Oct. 20, 1932, and was discharged on November 15. Thirteen years before he had attacks of cramplike pains in the upper part of the abdomen and diarrhea; the latter was never bloody. On the advice of a physician, he avoided raw fruit and vegetables in an effort to control the diarrhea. This dietary regimen had not been imposed long when swollen, painful gums which bled easily developed. The patient consulted several doctors and dentists regarding this condition and obtained no relief until one of the physicians advised washing them with fruit juice. After a few weeks, the gingival condition improved greatly. Fearing, however, that the fruit juices and fresh vegetables would increase the severity of the diarrhea, the patient again eliminated these foodstuffs, with the result that there was a recurrence of the swollen, bleeding gums. The latter condition was again relieved by the ingestion of fruits and fresh vegetables, only to reappear when these foods were suspended. This order of events repeated itself several times. Finally, six months before admission the gums again became swollen and red. The patient had failed to profit by his past experience and was subsisting on a diet composed of cereals, meat, bread and uncooked greens. The diarrhea was apparently controlled by the medication which he had consistently taken for the past thirteen years.

For a few years he had had pains in the lower extremities. These took on a more definite character about one and one-half years before admission. At that time he began to complain of pains in the joints of the left lower extremity and in the right foot. These distressing attacks were made worse with the advent of damp weather.

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2. Frazier, C. N., and Hu, C. K.: Cutaneous Lesions Associated with Deficiency in Vitamin A in Man, Arch. Int. Med. **48**:507 (Sept.) 1931.

On admission, he presented pallor of the skin and mucous membranes. There were dull red, fleshy, follicular papules scattered over the thighs and legs (fig. 1). Each lesion was pierced centrally by a hair which was either curled up or broken off and surrounded by an accumulation of hyperkeratotic material. The teeth were in poor condition. The gums were swollen, red and turgid and bled easily (fig. 2). The medial aspect of the left knee was slightly tender on palpation. The blood pressure was 200 systolic and 126 diastolic. The hemoglobin was 92 per cent; the white blood cell count was normal. The platelets numbered 200,000. The bleeding time was three minutes; the coagulation time, seven and one-half minutes. The tourniquet test, performed on the right arm, gave strikingly positive

Fig. 1.—The follicular papules of scurvy on the lower part of the thighs and the upper part of the legs.

results, revealing a peculiar feature which merits emphasis. On the right forearm, numerous tiny hemorrhages appeared which extended to the wrist and were confined mainly to the follicles of the hair in this region.

On measurement of the legs, atrophy of the left leg was noted. The left ankle jerk was slightly diminished. There were no sensory changes. The neurologist suggested toxic involvement of the anterior roots of the left side as the cause of this condition. The orthopedist could find no diseases of the bones or joints to account for the symptoms in the left lower extremity. The Rehfuss test showed normal values, and the roentgenographic studies revealed no evidence of an organic lesion in the gastro-intestinal tract. Roentgenograms showed no evidence of a subperiosteal hematoma.

In addition to the regular diet, the patient was given 24 ounces (709 cc.) of orange juice each day. In the course of one week, he had so strikingly improved that the orange juice was reduced to 16 ounces (473 cc.). The gingival swelling and bleeding disappeared, the tourniquet test became negative, and the pain in the left leg diminished to such an extent that the patient became ambulatory. The cutaneous lesions, some of which had in the interim assumed a hemorrhagic char-

Fig. 2.—The swollen, turgid gums of scurvy.

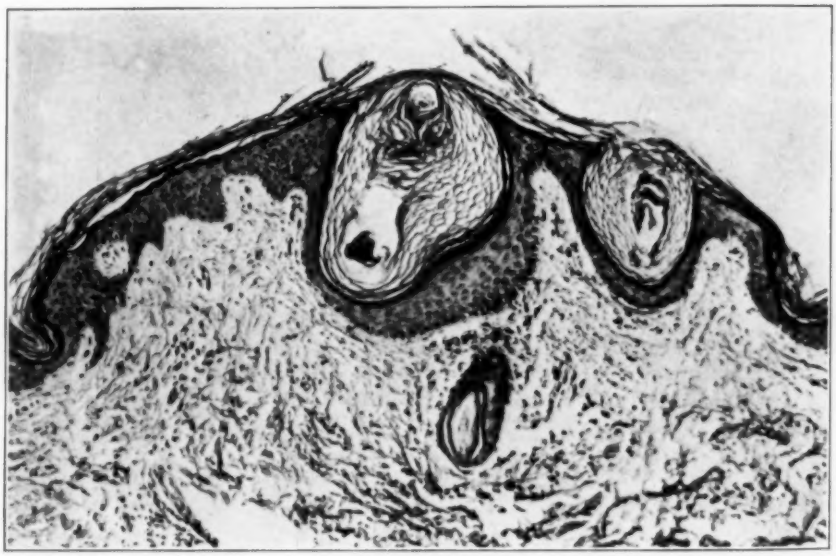

Fig. 3.—The entire papule with its central hair follicle containing a hyperkeratotic mass. The intense edema is clearly seen. The capillary congestion and the moderate interstitial hemorrhage cannot be seen.

acter, began to fade, leaving several small spots of brownish pigmentation before completely disappearing.

A biopsy of one of the follicular lesions on the anterior aspect of the left thigh was performed on October 24. The histologic observations were of interest since they helped to explain the clinical variations of the eruption.

The photomicrograph (fig. 3) demonstrates most of the essential features. The entire papule is shown with two adjacent hair follicles piercing it centrally and

containing curled hyperkeratotic masses. Just below, there is a cross-section of a hair which on serial section leads into one of the hair follicles. The principal changes are seen in the upper cutis in relation to the follicular and perifollicular regions. In this zone there is an intense edema causing separation of the collagen bundles without, however, obvious degeneration of the latter in hematoxylin-eosin stain. The capillaries show fragmentation with occasional thickenings of the walls, and in general their outlines are difficult to distinguish. There are a few collections of endothelial cells and cells resembling those derived from the reticulum. There is a definite tendency of the red blood corpuscles to be perivascularly grouped around apparently intact capillaries. Most of the red blood cells are, however, scattered interstitially. In the adjoining perifollicular region there are many small, dilated capillaries filled with red blood cells. The capillaries seem to possess intact walls, although scattered red blood corpuscles may also be found loose in the surrounding tissues.

The presence of the intense edema and to a lesser extent the slight proliferation of the fixed connective tissue cells explain the mechanism by which the follicular and perifollicular lesions assume a papular character. The exact nature of the vascular pathologic changes cannot be judged from the study of a single case. There was no evidence of acute or chronic inflammation.

Elastica and other stains were unfortunately not used.

Comment.—The lesions in this case were dull red, follicular papules, distinctly fleshy or urticarial on palpation. Each lesion bore a deformed hair surrounded by a cone of hyperkeratotic material. The end-stage was represented by the perifollicular hemorrhagic eruption produced by the application of the tourniquet to the right arm.

The pathologic changes in the biopsy specimen from the thigh fully explain the clinical character of the eruption. The follicular hyperkeratosis obviously corresponds to the identical gross appearance. The intense edema practically accounts for the raised, urticaria-like character of the dermatosis. The dull red color is explained by the capillary congestion associated with the presence of red blood cells in the interstitial tissue. Although we have no biopsy to prove the point, it would appear that the characteristic petechial lesion of scurvy depends on a more advanced degree of capillary damage, resulting in either rupture or more intense diapedesis. That the eruption disappeared under an antiscorbutic diet at a time when the tourniquet test became negative is worthy of note.

REVIEW OF THE LITERATURE

The subject of follicular lesions resulting from deficiency diseases has narrowed itself down to a consideration of scurvy and vitamin A deficiency. The cutaneous lesion of scurvy was known to older clinicians; that in vitamin A deficiency is a recent addition to medical knowledge. A consideration of these dermatoses is complicated by the fact that several vitamin deficiencies not infrequently coexist.

While the perifollicular petechial eruption of scurvy characterizes the more severe instances of the disease, it occasionally happens that obvious hemorrhage is absent, the follicular changes manifesting themselves clinically as the so-called "scorbutic gooseflesh." The older clinicians (Jessner [3]) spoke of it as "lichen scorbuticus," at a time when the term lichen indicated any type of papule. Studies on a more extensive scale were rendered possible by the World War, several reports appearing almost simultaneously.

In 1918, Nicolau [4] described follicular papules in scurvy as an original observation. He recognized the primary lesion as a minute horny papule due to intrafollicular keratosis. Perifollicular infiltration of a chronic torpid type constituted the end-stage. In the majority of instances, the eruption began almost simultaneously on the forearms and thighs and occasionally in the subumbilical region on each side. The eruption was characterized by a remarkable symmetry. In mild cases it remained localized to the aforementioned sites, but in severe cases it spread progressively to practically all of the hairy regions of the body except the scalp. There were no subjective complaints referable to the dermatosis. Nicolau mentioned the fact that under adequate treatment the lesions eventually disappeared.

In 1919, Wiltshire [5] described his experiences based on three thousand cases of scurvy observed in the Serbian army. In a series of seventy-eight consecutive cases, he found only 4 per cent without involvement of the hair follicles. In 87 per cent of the cases, there was follicular hyperkeratosis either alone or in conjunction with perifollicular hemorrhage. The favorite sites of implication were the anterior and inner aspects of the thighs and the upper parts of the legs, as in our two cases. Occasionally the lesions were so numerous that practically every hair follicle in these areas was affected. In many instances, the pubic hair was also involved (20 per cent), and less commonly the hairs on the back of the wrists and forearms (2.5 per cent). Wiltshire stated that each affected hair follicle showed a hard concentric swelling, pinhead in size, resulting from a collection of horny epithelial débris accumulated in the mouth of the follicle. In many, there was a thin atrophic or broken hair stump projecting from the summit of the cone; in others, the hair was wanting, having been shed or broken off flush with the surface. Several weeks after recovery from the disease, the hair grew again. Permanent loss of hair did not seem to occur. Wiltshire considered this an early sign of great

3. Jessner, S.: Haut Anomalien bei inneren Krankheiten, Berlin, A. Hirschwald, 1893.

4. Nicolau, S.: Ann. de dermat. et syph. **7**:399, 1918.

5. Wiltshire, H.: Lancet **2**:564, 1919.

importance, and any soldier showing such follicular papules was first put on an antiscorbutic diet before operation. In common with Wiltshire, we do not believe that such lesions are due to simple wasting, for we have not seen lesions of this sort in persons suffering from cancerous cachexia.

In 1919, Aschoff and Koch [6] described similar lesions, emphasizing the fact that the passage of the hand across affected areas of this sort produced the impression of "goose flesh." The hairs were easily broken and assumed a corkscrew shape or they were entirely lacking. The addition of hemorrhage resulted in a more conspicuous lesion.

In 1931, Frazier and Hu [2] reported instances of vitamin A deficiency in fifteen Chinese soldiers, all of whom suffered from keratomalacia. Several weeks prior to the onset of ocular symptoms, the skin became dry and slightly rough. Subsequently spinous processes developed at the site of the hair follicles, especially those on the anterolateral aspect of the thighs and the posterolateral aspect of the upper part of the forearms. Gradually this condition spread to other hairy portions of the body. The follicular papules varied in size according to the stage of development and the degree of perifollicular infiltration. The largest papule was about 0.5 cm. in diameter, hemispherical and firm; it was usually surrounded by a zone of pigmentation and held a keratotic plug at its apex in the form of either a hard spinous process or a loosely adherent scale. There were no clinical signs of scurvy, pellagra or beri-beri. On a balanced diet combined with cod liver oil and one lemon a day, there was immediate and striking improvement in the ocular and cutaneous manifestations. The keratotic papules decreased in size, and many of the central cornified plugs were extruded. Delicate atrophic scars remained at the site of many healed lesions and were surrounded by a zone of pigmentation. In five cases there was a marked tendency to pustulation, especially in the lesions on the extremities. This resulted in the formation of ecthymatous ulcers which, as a rule, were only few in number except in one instance in which the ulcers were so widespread as to suggest secondary ulcerative syphilis. The histologic study revealed hyperkeratosis of the epithelium of the epidermis and the hair follicles. The mouths of the hair follicles were greatly dilated by dense masses of horny substance. Coiled atrophic hairs were found in some of the follicular plugs. The lower parts of the hair follicles were atrophic. Pustulation occurred primarily in the follicular plugs and secondarily extended into the perifollicular tissues. Hemorrhage was not present. Frazier and Hu's conception of the pathologic process in vitamin A deficiency is interesting. Accord-

6. Aschoff, L., and Koch, W.: Veröffentlichungen aus d. geb. d. Kriegs- und Konstitutionspathologie, Jena, Gustav Fisher, 1919.

ing to the evidence at hand, it would appear that the skin and ocular manifestations are produced in a similar manner. In both, the primary process appears to be an excessive epithelial keratinization, leading in one case to xerophthalmia and in the other to the spiny follicular papules. As a result of the decreased resistance of the tissues, secondary infection takes place, which results on the one hand in corneal ulcers and on the other hand in cutaneous ulcerations.

Loewenthal [1] recently described the cutaneous manifestations of vitamin A deficiency, his report being based on one hundred and thirty cases of a papulofollicular dermatosis associated with xerophthalmia and night blindness. In the main, his description tallies with that of Frazier and Hu except that Loewenthal observed no instances of secondary infection of the skin. It appears that his patients were suffering solely from vitamin A deficiency, whereas in the cases recorded by Frazier and Hu the patients in some instances at least not only were lacking in vitamin A but were generally undernourished. In Loewenthal's detailed histologic report, the epithelial pathologic changes are stressed, and the changes in the cutis are regarded as insignificant and secondary.

According to Frazier and Hu, the follicular lesions in scurvy are similar in morphologic appearance to those encountered in vitamin A deficiency. They emphasize the point that single dietary faults are uncommon. The implication is that the lesions of scurvy are possibly due to a coincidental lack of vitamin A, a view to which we cannot subscribe, for in the detailed reports on scurvy the other evidences of vitamin A deficiency were absent. From the descriptions at hand, it would appear that clinical similarities between the follicular lesions of these two diseases exist only in the earliest lesion of scurvy. When, however, the scorbutic process in the hair follicles is more advanced, striking differences arise, and it is for this reason that we wish to separate the two lesions. There are, however, other considerations which influence us to regard the processes as basically different.

In vitamin A deficiency, the fundamental pathologic change seems to be in the epithelium, resulting in hyperkeratosis of the epidermis and of the hair follicles. The lesion is not accompanied by inflammatory changes in the cutis (except secondarily), and, most important of all, hemorrhage does not seem to occur. Ulceration appears to depend on the lowered resistance of the tissues in patients who are undernourished. On the other hand, the fundamental process in scurvy resides in the capillaries of the upper cutis in the region of the hair follicles. As a secondary result, the hyperkeratosis is mainly limited to the hair follicles. Early in the process, hemorrhage of a minor degree occurs interstitially. This is, however, not always clinically discernible. In

our second case, the dull red color of the papule was explained by the congestion in the perifollicular capillaries and the moderate interstitial hemorrhage. When the hemorrhage becomes sufficiently intense, the typical perifollicular petechia of scurvy results. This was artificially accomplished in our second case by means of the tourniquet test, in which the maximal capillary fragility was demonstrated to be almost entirely confined to the perifollicular vasculature. The early occurrence of a positive tourniquet test which becomes negative when the disease is properly treated illustrates the point that vascular damage is the fundamental pathologic process in scurvy.

CONCLUSIONS

1. Follicular lesions occur in both vitamin A deficiency and scurvy.

2. The earliest lesion of scurvy appears to be clinically indistinguishable from that observed in vitamin A deficiency.

3. The advanced follicular lesion of scurvy possesses characteristics which permit differentiation from that seen in vitamin A deficiency.

4. While both diseases attack the hair follicles, the fundamental processes appear to be different. In vitamin A deficiency, the chief effect is on the epithelium of the epidermis and of the hair follicles. Vascular pathologic change is insignificant, and hemorrhage does not seem to occur. In scurvy, however, the vascular factor is the most prominent feature, and it is this which serves to produce the distinguishing characters.

509 Madison Avenue.

Mr. A. Lowenthal permitted us to use his photographs and Dr. S. Otani, the photomicrograph.

PIGMENTED MYCOTIC GROWTH BENEATH THE NAIL

WILLIAM J. YOUNG, M.D.
LOUISVILLE, KY.

The subject of diseases of the nails has always been preplexing. The term onychia or paronychia has often proved a blessing in disguise until the true nature of an infection was elicited. With the avalanche of literature concerning mycotic infections, the task of diagnosis has become easier. To further the diagnosis of diseases of the nail I present the following report.

REPORT OF A CASE

A. R., a white man, 38 years of age, a dentist, sought advice regarding a painful nail with a black linear growth, ⅛ inch (0.32 cm.) wide, extending from the matrix to the end of the nail of the third finger on the right hand (fig. 1).

The lesion had made its appearance two years before as a red streak, advancing from the matrix with the growth of the nail. Simultaneously, there was a gradual formation of a ridgelike convex elevation of the entire nail. Approximately one year later a black spot appeared in the matrix at the base of the red streak. The black spot gradually enlarged and progressed to the distal end of the nail. This produced a straight black line, as shown in figure 1. Pain was produced by pressure over the darkened ridge and at the tip of the nail. The pain became progressively more severe and constant. When the patient was first seen the finger was excruciatingly painful.

On examination a black growth was found underneath the nail which could be broken off piecemeal, independent of the nail itself. The patient remarked that the black structure grew faster than the nail and had to be trimmed more frequently than the nail.

Particles of the growth were cultured and the following observations made (report of Dr. Clyde McNeill): Cultures of the black pigmented material from beneath the nail were observed after five or six days (fig. 2). The growth which spread over the surface of the agar slant was grayish with occasional patches of white (fig. 3). The white patches were due to sterile mycelium. Later, a dense black pigment diffused throughout the medium. Transplants were obtained by the slide culture method, and transfers were made from a single spore to maturity. The chief characteristics were: a rich growth in Sabouraud's maltose agar with a black substratum; vegetative mycelium with numerous cross-walls; spores not borne in spore case; conidiophores not united into definite bodies; smoky-colored hyphae and conidia; conidia of more than two cells, containing cross-septums but no longitudinal septums; conidia in terminal heads; systematic position, Dematiaceae Macronemeae Acrothecieae; identification, Acrothecium nigrum.

No treatment was given other than a series of holes bored through the ridge in the nail over the black growth. The pain gradually lessened and finally ceased. The black growth slowly disappeared by advancing distally, leaving normal tissue in its wake. It seemed reasonable to assume that normal growth of the nail

Fig. 1.—The nail as seen on the first visit. Note the relative width and the location of the black growth.

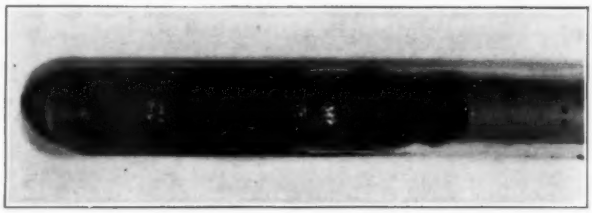

Fig. 2.—A tube culture (Sabouraud) showing the tiny colonies on the surface and the black pigment staining the agar after five or six days' growth.

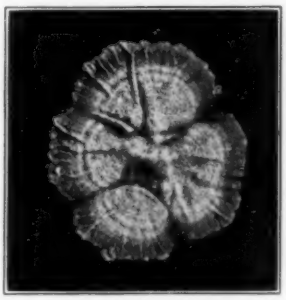

Fig. 3.—A typical colony appearing as a white or gray patch on the agar slant. It has been placed on a black surface for photographic contrast.

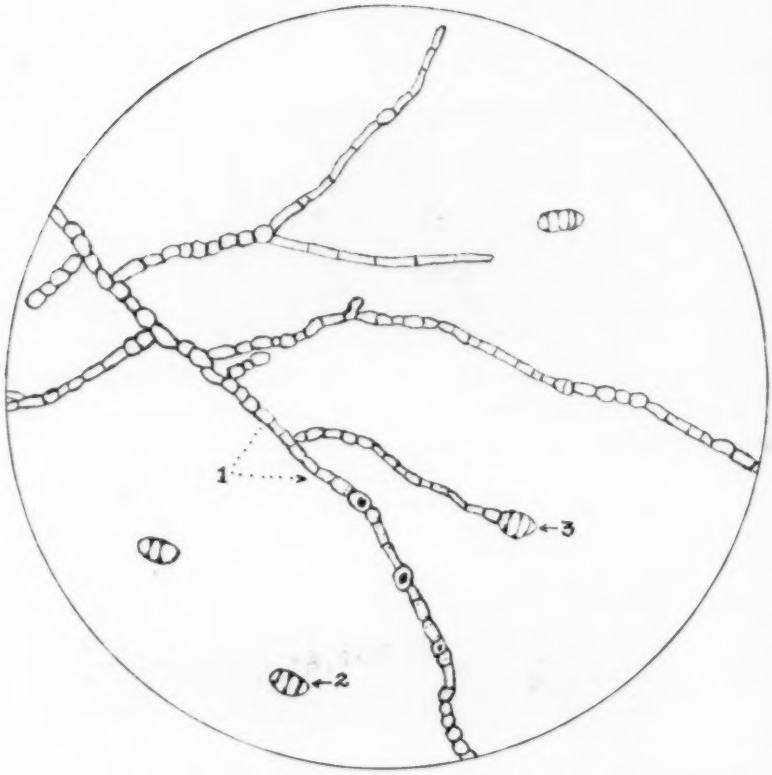

Fig. 4.—Camera lucida drawing showing the pigmented (*1*) hyphae, (*2*) cross-striated conidia and (*3*) terminal conidia; reduced from × 380.

Figure 5

Figure 6

Fig. 5.—A photograph taken after the nail had been pierced with a series of small holes over the pigmented growth. Note the thinning and fading of the pigment at its base in the matrix.

Fig. 6.—Normal appearance of the nail after the disappearance of the black pigment.

would be restored. It is interesting to note that the patient believed that the condition of the nail was due to a small apical abscess of the upper lateral tooth on the right side. The extraction of the tooth was coincidental with the healing of the nail.

CASES IN THE LITERATURE

In 1917, Montgomery [1] reported a condition in a Negress which he had never observed before and which he had never seen mentioned. A discolored band, 3 mm. broad, ran up the center of the thumb-nail from the base to the distal edge. It could be seen to lie in the whole thickness of the nail. The most reasonable explanation for the phenomenon seemed to be that it was the result of a pigmented nevus situated in the groove of the nail, producing pigment in the newly formed nail. No sections were made.

Templeton,[2] in 1926, described a case showing a dark longitudinal pigmented stripe of the finger-nail. A search of the available literature had disclosed but one similar case. The line began at the base and grew out to the tip of the nail. For seven years it was no wider than a hair. During the last two years it had slowly grown until it was about 2 mm. wide. Histologic section showed that the pigmentation extended throughout the entire thickness of the nail, the cornified cells being filled with a fine granular dark brown pigment. As the cells became flattened toward the outer surface the amount of pigment in them seemed to decrease, but some of the flat cells at the surface still contained a small amount of pigment. The pigment was considered to be melanin. Templeton put the lesion in the class of pigmented nevi. From the photographs his case appears to be identical with mine, but the pigmentation was in the nail proper, whereas in my case the nail and the black growth were separate entities. I feel that the conditions are distinctly different.

A case reported by Monash [3] showed a pigmented deposit along the lateral borders of the nails. There is apparently no similarity between this case and mine.

---

1. Montgomery, D. W.: A Pigmented Stripe in the Thumb Nail, J. Cutan. Dis. **35**:99, 1917.

2. Templeton, H. J.: Pigmented Stripe in the Nail, Arch. Dermat. & Syph. **14**:533 (Nov.) 1926.

3. Monash, Samuel: Normal Pigmentation in the Nails of the Negro, Arch. Dermat. & Syph. **25**:876 (May) 1932.

VALUE OF KLINE PRECIPITATION TEST FOR DETECTION OF SYPHILIS IN APPLICANTS FOR LIFE INSURANCE

CHARLES ROBERT REIN, M.D.
AND
MARGUERITE LE MOINE
NEW YORK

There has been an increasingly widespread use of the various precipitation tests for the diagnosis of syphilis, because these tests are simple in technic and are as accurate as the complement-fixation reactions. Of the various precipitation tests, the microscopic slide test developed by Kline [1] has been found by many to be very accurate and simple in technic. Littman [2] reviewed 147,250 Kline tests performed in 16 clinics in various parts of the country and found them consistent with the clinical findings in from 96.52 to 99.18 per cent of the cases. He found the Kline test to be more sensitive than the Kahn, Wassermann or Hinton tests in various stages and types of syphilis.

For years the Metropolitan Life Insurance Company has hoped for the development of a method for the detection of syphilis in which the blood could be obtained in small amounts from a simple puncture of the finger or of the lobe of the ear. The test would have to be one which would give satisfactory results with the specimen exposed to relatively high temperatures and would have to be stable for as long as ten days after collection, to allow ample time for the specimen to reach the laboratory of the home office from outlying districts. The Kline test seemed to meet these requirements, and it was decided to make a preliminary study [3] at the laboratory, small amounts of blood being used. Specimens were obtained from employees and from others examined at the home office.

In these cases a specimen of blood was collected by puncture of the finger. At the same time, a sufficient quantity of blood was drawn from the arm for Wassermann and Kahn tests. The subjects were divided into syphilitic and nonsyphilitic groups, 18 (16.3 per cent) of the specimens being obtained from persons who were known to have syphilis from previous blood tests and 92 from persons who had no syphilitic

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From the Laboratory Department, Metropolitan Life Insurance Company.

1. Kline, B. S.: Microscopic Slide Precipitation Tests for Syphilis, Baltimore, Williams & Wilkins Company, 1932.

2. Littman, S.: Clinical Evolution of 147,250 Microscopic Slide Precipitation Tests for Syphilis, Urol. & Cutan. Rev. **36**:245 (April) 1932.

3. Rein, C. R.: The Value of the Kline Precipitation Tests for Syphilis, Proc. A. Life Insur. M. Dir. America **19**:275, 1932.

history. In the 92 nonsyphilitic cases there was no disagreement between the results obtained with the Kline test and those obtained with the Wassermann test. In the 18 syphilitic cases there was disagreement in the results obtained with 2 of the specimens, both of which were positive with the Kline test and negative with the Wassermann test.

A comparison of the results obtained with the Kline and Kahn tests showed a 100 per cent agreement in the 92 nonsyphilitic cases, while there was disagreement in 1 of the 18 syphilitic cases, resulting in 98.1 per cent agreement in the syphilitic group. The specimen with which the disagreement occurred was positive with the Kline test and negative

TABLE 1.—*Comparison of the Results of Five Hundred and Ninety-Three Diagnostic Kline Tests and Wassermann Tests*

	Agreement	Relative Agreement	Disagreement: Positive Kline Tests Negative Wassermann Tests	Disagreement: Negative Kline Tests Positive Wassermann Tests	Total Tests
Tests	534	43	16	0	593
Per cent	90.05	7.25	2.69		
Tests	577		16		593
Per cent	97.30		2.69		

TABLE 2.—*Comparison of the Results of Six Hundred and One Diagnostic Kline Tests and Kahn Tests*

	Agreement	Relative Agreement	Disagreement: Positive Kline Tests Negative Kahn Tests	Disagreement: Negative Kline Tests Positive Kahn Tests	Total Tests
Tests	563	24	12	2	601
Per cent	93.66	3.99	1.98	0.33	
Tests	587		14		601
Per cent	97.67		2.32		

with the Kahn test. Thus, if only a Wassermann or a Kahn test had been done, these cases of syphilis would have been missed.

There were no false positive reactions obtained by any of the methods in the nonsyphilitic group, resulting in a 100 per cent agreement.

In a further study, there were 980 cases in which two or more tests were done. Kline and Wassermann tests were performed in 593 of these cases and Kline and Kahn tests in 601.

RESULTS

The results obtained with 593 diagnostic Kline tests and Wassermann tests are compared in table 1, and those obtained with 601 diagnostic Kline tests and Kahn tests in table 2.

METHOD OF PROCEDURE

The field examiner is requested to obtain a specimen of blood in the following manner: A mailing container is sent to him inside of which is a cloth jacket fitted with pockets. In these pockets are placed two glass pipets stoppered at both ends with "rubber policemen" (a small length of rubber tubing closed at one end) and a straight no. 5 Hagedorn needle for making punctures. The pipets are 4½ inches (11.4 cm.) long and 4 mm. on the inside diameter and are made from ordinary glass tubing, tapered slightly at one end (figs. 1 and 2). This outfit is accompanied with detailed directions for the collection of the specimen. One of the pipets should be filled two thirds full of blood and returned with the outfit to

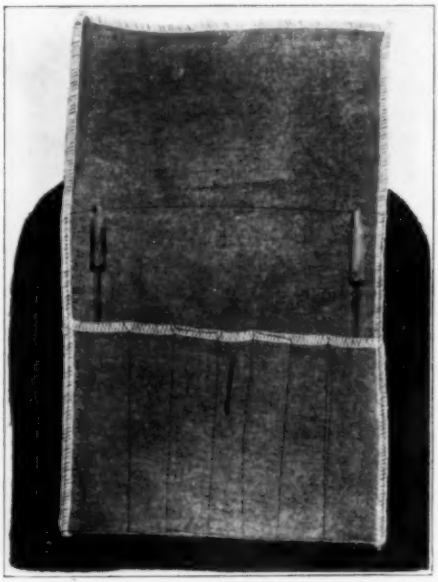

Figure 1

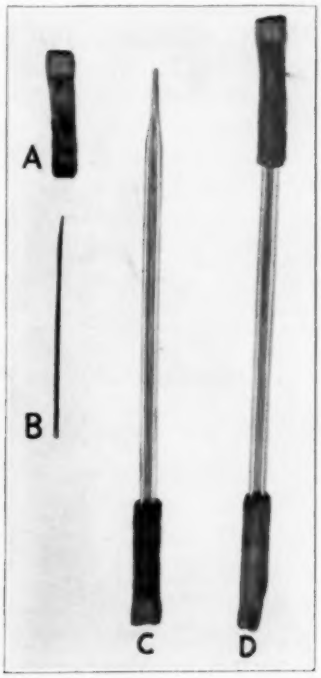

Figure 2

Fig. 1.—Outfit as received by the field examiner.

Fig. 2.—*A*, "rubber policeman"; *B*, no. 5 Hagedorn needle; *C*, pipet, and *D*, pipet stoppered with "rubber policemen."

the home office. The amount of serum obtained from this quantity of blood provides sufficient material for at least two Kline tests, so that if there is any doubt regarding the result, the test can be repeated immediately.

On arrival at the laboratory, the pipet is labeled with the name of the applicant, the "rubber policemen" are removed, and the tapered end of the pipet is sealed in a flame. Then a fine wire is introduced into the open end of the pipet containing blood, and the clot is broken. Several specimens tubes are wrapped in a paper towel and placed in a centrifuge cup and centrifugated for one-half hour at high

speed after which the tubes are immersed in a water bath at 56 C. for one-half hour, thus inactivating the specimens of blood.

After inactivation, a file mark is made on the pipet containing the blood just above the point where the serum and clot meet, and the pipet is broken with the fingers. The serum is delivered into a graduated 1 cc. pipet by holding the point of the graduated pipet to the broken end of the pipet containing blood. The graduated pipet is held at a downward angle, and the serum usually flows into it, though at times it may be necessary to apply suction.

The preparation of the Kline diagnostic antigen emulsion, the method of mixing the latter with the serum and the interpretation of the test are described in detail by Kline.[1]

COMMENT

Hazen [4] estimated that about 400,000 cases of fresh syphilis develop each year in the United States, but that only about 42 per cent of the infected persons undergo treatment during the first twelve months. The majority of the remaining persons in time become free from the cutaneous manifestations and constitute the group with latent or concealed syphilis. The incidence of syphilis necessarily varies with the race and social strata of the population.

For obvious reasons, taking a venereal history is practically useless in an examination for life insurance. Many persons with latent syphilis do not know that they have been infected, and the majority of those who know they have or have had the disease deny it. Thus, many of these syphilitic persons pass the examination, because it is not always possible to recognize syphilis in the latent stage. That this is not merely a conjecture is proved by the following: Work with various transfusion boards in which routine blood tests for syphilis were made revealed a surprisingly large number of professional donors with concealed syphilis, who volunteered the information that they had recently passed an examination for insurance. It is reasonable to expect that a certain number of those seeking insurance have syphilis in the latent form which, in many instances, would be detected only by an examination of the blood.

Because there is a greater morbidity among untreated syphilitic persons, it is clear that diagnosis and treatment of the disease are imperative. This is of special importance to insurance companies, and any test that lends itself to easy diagnostic use must prove invaluable. The Kline test seems ideal for the purpose.

Schamberg [5] pointed out that it may be worth while for life insurance companies to require a blood test for the detection of syphilis precedent to the issuance of policies. He stated that this would lessen the cost of premiums to nonsyphilitic policy holders, would serve as an important

4. Hazen, H. H.: Treatment of Cardiovascular Syphilis, Am. J. Syph. **16**:289, 1932.

5. Schamberg, J. F., and Wright, C. S.: Treatment of Syphilis, New York, D. Appleton and Company, 1932.

educational program, would make available valuable statistical data on the prevalence of syphilis and would reveal to thousands of people the fact that they are suffering from syphilis at a period when it can be treated successfully.

CONCLUSIONS

The Kline test for syphilis which is particularly useful for the detection of syphilis in applicants for insurance is described:

1. The blood is easily obtainable from a finger puncture.

2. Only a small amount of blood is required.

3. No chemicals need be added to prevent clotting or bacterial contamination.

4. The specimens can be sent with safety over long distances to a central laboratory for examination.

5. The specimens are satisfactory for testing even ten days after collection.

6. This test appears to be more sensitive and more accurate than the Wassermann and Kahn tests.

A RARE SWEAT GLAND TUMOR

SYRINGOCYSTADENOMA NODULARIS

RICHARD L. SUTTON JR., M.D
KANSAS CITY, MO.

An unusual case recently came to my notice in the practice of Dr. Richard L. Sutton Sr.

REPORT OF A CASE

History.—Mrs. W. L. H., a white woman, 45 years of age, a housewife, presented herself complaining of lumps in the skin of her neck. The nodules were grouped on the right side of the neck and scattered in a band extending from the vertebra prominens to the region of the sternoclavicular articulation (fig. 1). They had been present since childhood, and were slowly enlarging. A few new ones were appearing as flattened ovoid tumors about 2 mm. in diameter. The only symptom was slight tenderness on pressure, and the reasons for requesting examination were that new lesions were appearing and that the condition was unsightly.

On examination the lesions were found to be relatively soft and elastic, and not palpably cystic. They were rounded and apparently encapsulated, discrete nodules from 2 to 10 mm. in diameter. They moved freely over the deep fascia but were fixed to the overlying skin, which was slightly thinned, glazed and telangiectatic over the largest ones. In the group of larger nodules each rounded tumor could be palpated separately. Lower on the neck and over the jugular notch they were smaller and discrete. The distribution was strictly unilateral, lying roughly in a zone extending from a group over the vertebra prominens to the jugular notch. The regional lymph glands were unaffected; the general condition was normal. Other lymphatic regions, the spleen and liver, the urine and the white blood cell and differential counts were normal. The patient's general health was not affected.

In the course of a few weeks, several nodules, including the large group, were excised and examined microscopically. The wounds, closed with catgut, healed by primary intention, and the cosmetic effect was satisfactory. There was no recurrence in a year at the sites of excision.

Dr. H. R. Wahl, head of the department of pathology of the University of Kansas Medical School, examined some of the tissue, which was presented to him with the clinical diagnosis of syringocystadenoma nodularis, and reported as follows:

Gross Examination.—The specimen consisted of a pyramid-shaped portion of skin and subcutaneous tissue measuring 2 by 1.6 by 1 cm. The skin was smooth, and there was one nodular elevation of the cutaneous surface measuring 9 mm. in diameter. The nodule was rubbery, and on section formed a portion of a larger subcutaneous ovoid structure which was well encapsulated and had a smooth grayish-white cut surface mottled by small focal hemorrhages.

Histologic Examination.—The section showed a cellular and fairly well encapsulated nodule of tissue just under the epidermis. In some places small clusters of

mucous-like glands could be seen here and there with cylindric epithelial cells and nuclei toward the base. The bulk of the tissue, however, was composed of a cellular growth in which the cells were deep staining and contained a small amount of cytoplasm. Frequently they seem to be arranged in solid nests, occasionally with cleftlike spaces between the individual cells. Little cytoplasm could be recognized around the nuclei. Some variation in nuclear type was noted, but this was not striking. The tissue did not appear to be malignant. A tendency for the cells to form acini was suggested in many places. There was a considerable number of what appeared to be lymph spaces. The tumor cells did not rest directly on the endothelial lining, but were usually separated by a small amount of acidophilic tissue. In some places the stroma was loose and edematous and showed some hemorrhages. In most places it was scanty.

Fig. 1.—These discrete, elastic nodules in a bandlike distribution had been present on the patient's neck since childhood. They were enlarging slowly; a few new ones were appearing, and they were tender on pressure.

Between the capsule of the tumor nodule and the epidermis were some flattened bodies which contained a few scattered elongated epithelial cells and a few dilated cystic spaces, evidently derived from sweat glands.

The diagnosis was syringocystadenoma nodularis.

In my own microscopic examination of sections of three different nodules, several additional observations were made. The cellular proliferations seemed to take the form of cylinders, branching, massed and interlaced and of varying diameter depending on the degree of proliferation of the peripheral layer of a tubule of two layers. The cells of this outer layer formed the mass of the neoplasm, and were uniform in size and structure. Their cytoplasm was scanty, and the nuclei were oval and deeply basophilic. They somewhat resembled lymphoid cells, as in lymphosarcoma. They were arranged in bands and loose alveoli to a varying thickness about small tubules composed of a distinctly different type of

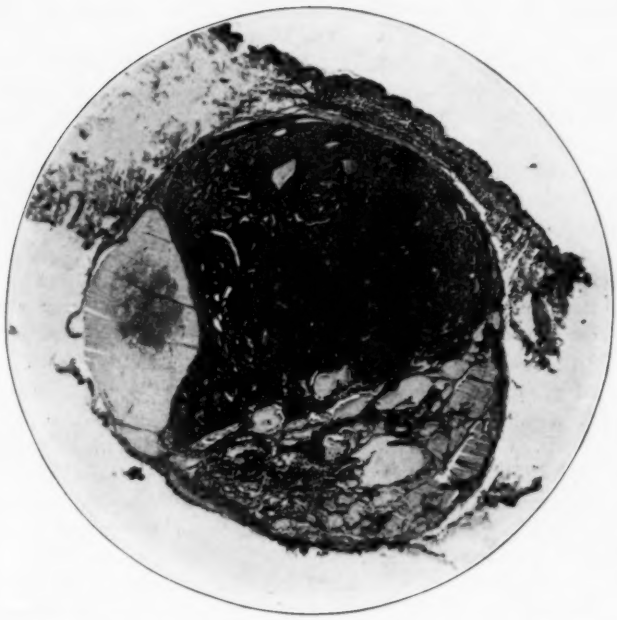

Fig. 2.—Section of a nodule, showing its relation to the overlying skin, the definite encapsulation, the general topography and the cystic structure; × 10.

Fig. 3.—The cellular proliferations seem to take the form of cylinders, massed and interlaced and of varying diameter, depending on the degree of proliferation of the peripheral layer of a tubule of two epithelial layers; × 80.

cells. These central groups cut across bore a resemblance to the proximal convoluted portion of renal tubules, in that the epithelium was a single layer, the nuclei were pale, and the cytoplasm was pink-staining (hematoxylin and eosin). While this structure was clearly evident in parts, more complexly proliferating portions were commoner. My interpretation of the architectonic scheme was that the tumor growth tended to produce tubules surrounded by proliferating cells resembling lymphoid cells. This is shown in the photomicrographs of the structure of the ducts and of the looser cylindric parts of one of the nodules. In consideration of this two-layered epithelial tubular structure I believe that the tumors had their origin in sweat glands. Weidman and Besancon[1] saw a "long, continuous double row of cells" in the syringomatous lesion of the shoulder in their case, and used this observation as evidence of origin in sweat glands.

The two types of cells, those resembling lymphoid cells and those resembling renal tubules, were in evidence in the more anaplastic nodule, as shown in the photomicrographs. Complex as this structure was the dark nuclei with little cytoplasm surrounded the paler, more vesicular cells.

The isolated groups of pale, foamy cells seen in the complex, anaplastic nodule, were in definite alveolar arrangement. These were interpreted as representing degeneration in cells resembling renal tubules, analogous to those observed in tumors of the breast by Dawson,[2] who interpreted similarly the pale cells occurring there.

In one of the nodules, as seen in the figures, the formation of cystic spaces was obvious. These spaces were lined by cells of the secretory type, which were flattened and more or less deteriorated. The content of the cyst was highly proteinous, for the precipitate looked solid, did not contract much and was granular and palely acidophilic. It was considered the product of the secretory alveoli of the tumor.

There was almost no intranodular stroma. That which was present was delicate and distended with the fluid injected for local anesthesia. The distortion and indistinctness of the secretary alveoli, in many places compressed almost to obliteration by the active proliferation of sheath cells, in places caused resemblance to a stroma. The arrangement of dark-staining sheath cells about these compressed pink strands might lead to misintepretation. It is on this basis, I presume, that the tumor has been looked on as endothelioma or lymphangioma, instead of a neoplasm of the sweat glands.

In the capsule of one of the nodules were isolated, fibrotic lobules composed of minute, cystic alveoli lined with low flattened cells within a dense fibrous stroma. The stroma had compressed some acini almost to obliteration. The lobules as a whole were distorted by the pressure of the large nodule, to the capsule of which they were adjacent. These I believe were separate anomalous adenomas of the sweat glands, representing individual sudoriparous anlagen—an opinion consonant with that of Weidman and Besancon[1] in their case. The neoplastic influence affected a number of embryonal sweat projects, but in different degrees; and these small, fibrotic syringomas were like papyraceous fetuses, abortive, fibrotic and compressed beside the luxuriant, vigorous tumor abutting them.

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1. Weidman, F. D., and Besancon, J. H.: Histologic Differences in a Syringoma of the Face and Shoulder, Arch. Dermat. & Syph. **21**:279 (Feb.) 1930.

2. Dawson, E. K.: Sweat Gland Carcinoma of the Breast, Edinburgh M. J. **39**:409 (July) 1932.

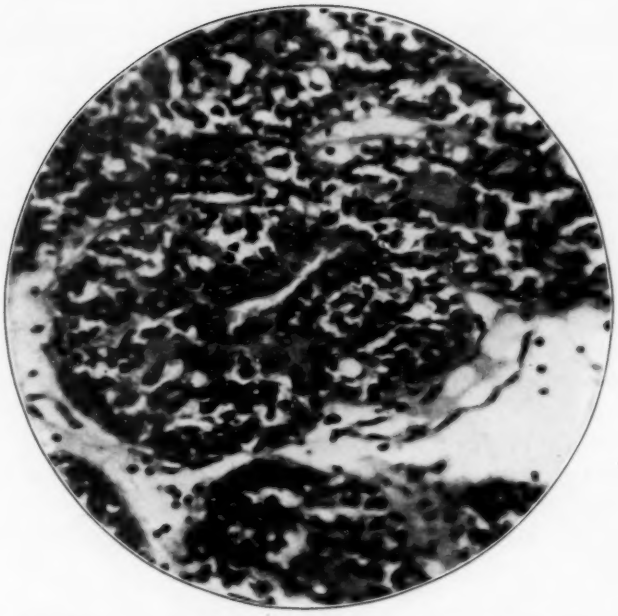

Fig. 4.—Proliferation is more active in the sheath cells, which surround derivatives of secretory cells in loose masses and alveoli. The stroma is scanty and delicate; the mass of the tumor is of epithelial origin; × 300.

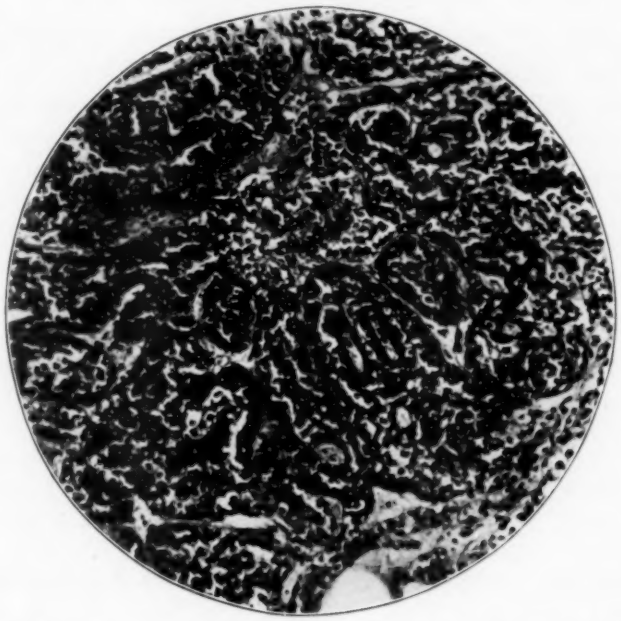

Fig. 5.—In the more complex parts the structure is still to be visualized as tubules surrounded by cells resembling lymphoid tubules, so that misinterpretation as endothelioma is possible; × 150.

COMMENT

In the simple classic syringoma (class 5, to be described) the proliferating, adenomatous element is the central layer of sweat cells. The peripheral layer is present, but has an attenuated, "stretched" appearance, as if it proliferated only sufficiently to keep pace with the more active growth of the secretory elements resembling renal tubules. Diametrically differing, the nodular tumors of this case presented a growth urge predominant rather in the layer of sheath cells, though also affecting the secretory tubules. To designate this fundamental difference, which I believe to be objectively true, and not a mere figment of interpretative imagination, I should call the nodular type represented by this case and by that of Paul and Adamson,[3] sheath cell syringocystadenoma while the classic syringocystadenoma is a secretory cell syringocystadenoma.

A comparable case was reported by S. Norman Paul and H. G. Adamson.[3]

> A man, 46 years of age, presented about twenty-five smooth, firm, raised growths on the chest on the left side of the sternum. The nodules were grouped, and their bases coalesced to form a vertically elongated plaque. The largest lesion was 2.5 cm. in diameter, and was raised 1.5 cm. above the general level; the smaller ones were of half the size of a pea. They had begun to appear twenty-five years previously, but new ones had presented themselves in the last three years.

Adamson said that they were not lobulated as in Brooke's[4] case. The tubular arrangement of cell growth suggested origin in the sweat glands rather than pilosebaceous origin. The cells presumably had lost their power of differentiation, but had retained their proliferative energy, as in basal cell tumors or rodent ulcer.

Paul and Adamson listed seven recognized types of cutaneous glandular neoplastic process: 1. Adenoma sebaceum. 2. Epithelioma adenoides cysticum of Brooke. 3. Multiple benign tumors of the scalp, the endothelioma of Spiegler[5] and Mulert, shown by Dubreuilh and Auché[6] to be benign epithelioma of basal cell origin. 4. Benign tumors of the lower orbital region with the structure of basal cell epithelioma of pilosebaceous or tricho-epithelioma type. 5. The type of probable

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3. Paul, S. Norman, and Adamson, H. G.: A Case of Syringoma, Brit. J. Dermat. **28**:106, 1916.

4. Brooke, H. G.: Epithelioma Adenodes Cysticum, Brit. J. Dermat. **4**:269, 1892.

5. Spiegler, E.: Ueber Endotheliome der Haut, Arch. f. Dermat. u. Syph. **50**: 163, 1899.

6. Dubreuilh and Auché: Epitheliomes bénins multiples du cuir chevelu, Ann. de dermat. et syph. **3**:545, 1902.

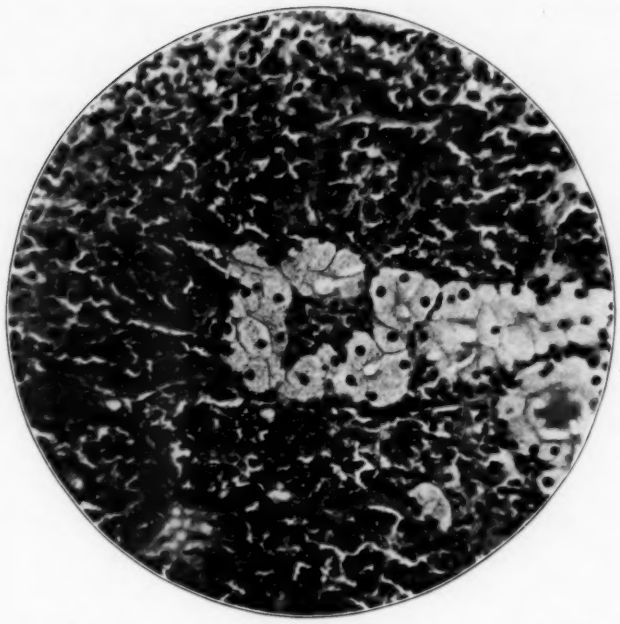

Fig. 6.—Isolated masses of foamy epithelial acini are present. These probably are degenerated tubules of secretory cells; × 300.

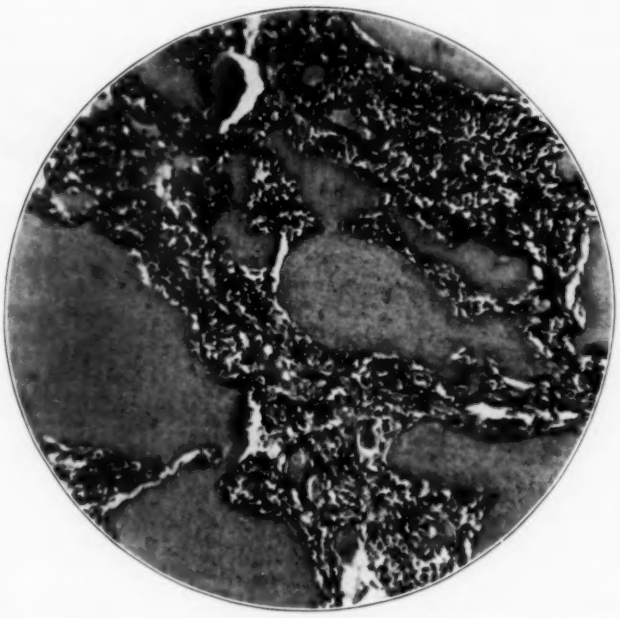

Fig. 7.—Cysts are present and filled with thick, coagulated, acidophilic protein. The content of the cysts appears to be derived from degenerate secretory cells; × 100.

origin in embryonic sweat glands described by Kaposi [7] as lymphangioma tuberosum multiplex, by Jacquet and Darier [8] as *hydradenome éruptif,* by Török [9] as syringocystadenoma, by Gassmann [10] as "naevi cysto-epitheliomatosi disseminata," and by Unna,[11] whose nomenclature is generally used, as syringoma or syringadenoma. This has been confused with the epithelioma adenoides cysticum of Brooke,[4] but its structure clearly recalls sweat gland rather than pilosebaceous apparatus. 6. Single tumors, the sweat gland or sudoriparous tumors or syringomas of Unna.[12] 7. Linear nevi made up of sweat glands or sebaceous glands.

Paul and Adamson's case was identical with one reported by Thimm,[13] whose report is inaccessible to me. Paul and Adamson thought that their case fell within the sixth group, as I should class the case reported here.

Paul and Adamson's list of types is fairly satisfactory. The characteristics of each type are well known despite their relative rarity.

1. In adenoma sebaceum the lesions range in size from that of a pinhead to that of a split pea, distributed over the face usually symmetrically. They appear generally in childhood or pubescence, and are benign and persitent. A hereditary tendency has been noted in rare instances, as in Shelmire's [14] family group of five.

2. Epithelioma adenoides cysticum of Brooke is equally well defined. It is not well called multiple benign cystic epithelioma, because this name is equally applicable to syringoma (Sutton and Dennie [15]). The lesions are small, firm and discrete, though rarely grouped and coalescent. They occur on the face, scalp, neck and chest. A familal incidence is frequently noted. The lesions sometimes harden, enlarge, ulcerate and become basal cell epitheliomas, as in cases noted by J. C.

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7. Hebra, F., and Kaposi, M.: Lehrbuch der Hautkrankheiten, Stuttgart, Ferdinand Enke, 1876, vol. 1, p. 282.

8. Jacquet and Darier: Hydradénomes eruptifs (épitheliomes adénoïdes des glandes sudoripares ou adénomes sudoripares), Ann. de dermat. et syph. **8**:317, 1887.

9. Török: Monatsh. f. prakt. Dermat. **14**:169, 1892.

10. Gassmann, A.: Fünf Fälle von Naevi cysto-epitheliomatosi disseminata, Arch. f. Dermat. u. Syph. **58**:177, 1901.

11. Unna, P. G.: Histopathology of Diseases of the Skin, translated by Norman Walker, New York, The Macmillan Company, 1896, p. 1117.

12. Unna,[1] p. 807.

13. Thimm, P.: Hypertrophie und multiple Cystadenome der Schweissdrüsen Knäuel, Arch. f. Dermat. u. Syph. **59**:3, 1904.

14. Shelmire, J.: Adenoma Sebaceum, J. A. M. A. **71**:963 (Sept. 21) 1918.

15. Sutton, R. L., and Dennie, C. C.: The Possible Interrelationship of Acanthoma Adenoides Cysticum (Multiple Benign Cystic Epithelioma) and Syringocystadenoma (Lymphangioma Tuberosum Multiplex), J. A. M. A. **58**:333 (Feb. 3) 1912.

Fig. 8.—In the capsule of one of the nodules is an isolated fibrotic cystic lobule, presumably an abnormal sweat anlage resembling the usual type of syringocystadenoma, in which stroma quantitatively exceeds epithelium; × 80.

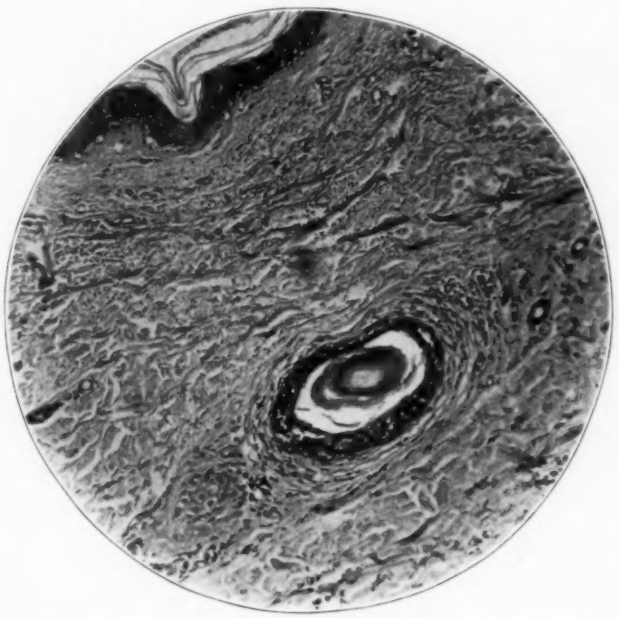

Fig. 9.—A duct, found in the zone intervening between the epidermis and capsule of one nodule. There is evident proliferation of sheath cells, and the laminated precipitate within the lumen resembles the accretions commonly seen in the usual form of syringocystadenoma; × 200.

White,[16] Stelwagon,[16] Jarisch,[16] Schamberg [16] and Sutton and Dennie.[16] Histologically one finds epithelial tubules of dense basophil cells resembling material of basal cell tumors; cross-sections of these tubules show a decided resemblance to hair follicles, and tumors developing from them are of the tricho-epitheliomatous type. In a case thoroughly studied, Weidman and Besancon [1] found epithelioma adenoides cysticum on the face and syringoma on the shoulder. They quoted Fox as remarking that epithelioma adenoides cysticum generally occurs on the face and syringoma on the trunk. In Sutton and Dennie's [15] report of two cases for the purpose of comparing epithelioma adenoides cysticum and syringocystadenoma, they noted that, "Both are multiple benign cystic epithelioma," and stated that it is better to confine this name to the Brooke-Fordyce type.

3. The multiple benign tumors of the scalp are peculiar benign epitheliomas. In structure they bear no small similarity to nodular syringomas, appearing as coiled tubules with proliferative walls set in the dense stroma of the connective tissue of the scalp. Adamson [3] felt that they are related to multiple benign cystic epithelioma. Whatever their relationships their clinical aspects are clearcut, and cases of firm, rounded multiple tumors in the scalp form a distinctive group not easily confused with other entities.

4. The benign tumors of the lower orbital region of tricho-epitheliomatous structure are, in my opinion, basal cell epitheliomas of a slow rate of growth. I have sectioned many waxy tumors removed from the upper half of the face, and they have shown remarkable variability as to minute structure, but always recalled either tricho-epithelioma or classic rodent ulcer.

5. The fifth group is the classic syringoma, or syringocystadenoma. The lesions are small, the size of a split pea, soft, superficial and often the color of chamois, and occur generally on the upper two thirds of the trunk, as in Finnerud's [17] case. The incidence is greater in women. The lesions appear first in adolescence, probably as a result of the general stimulation of the pilosebaceous and apocrine apparatus (Kyrle [18]). The name syringomacystadenoma was first used by Török [19] in 1889; Unna's names, syringoma or syringadenoma, have been generally used. The discrete, soft, little tumors are probably solitary adenomas of sweat glands, representing single anlage anomalies, according to Weidman and Besancon.[1] They are histologically typical, consisting of loosely

16. Quoted by Sutton, R. L.: Diseases of the Skin, ed. 8, St. Louis, C. V. Mosby Company, 1932, p. 723.

17. Finnerud, Clark W.: M. Clin. North America **14**:1147 (March) 1931.

18. Kyrle, quoted by Rosenberg, W. A.: Syringoma Influenced by X-Ray Therapy, Urol. & Cutan. Rev. **37**:295 (May) 1933.

19. Török: Monatsh. f. prakt. Dermat. **8**:116, 1889.

winding tubules of epithelium in a double layer, in concentrically disposed fibrous tissue. The lumens are often wide and frequently full of laminated or hyalinized accretion; but they may be compressed. In these curious little tumors the mesodermal portion, quantitatively, greatly exceeds the ectodermal. Most authors agree that they are adenomas (Weidman and Besancon,[1] Sutton and Dennie,[15] Unna [11] and C. J. White [20]), and the growth urge appears to lie in the secretory portion.

6. The single tumors, tumors of sweat glands or syringomas of Unna,[12] form an equally distinct group. In these the lesions are solitary or relatively few in number, and it is in this group that Paul and Adamson placed their case which is similar to the one reported here. The cases of this type which Unna described are few and unaccompanied by illustration. He wrote:

> The oldest reliable case of independent spiradenoma was published in 1859 by Lotzbeck, from von Bruns' Klinik. . . . It was a rapidly growing tumor on the cheek of a child, was half the size of a hen's egg. . . . Teased preparations showed that the tumor consisted largely of bent canals, as thick as the largest sweat glands, which showed spindle dilatations and constrictions between. . . . Lotzbeck found all the canals, with their appendages, clothed with cylindric epithelium. . . . In addition to the epithelia, there were in the canals numerous small, pale cells, like lymph corpuscles.

Another case was that of Thierfelder, in a woman of 50 years, "in whom four years previously, a soft, movable tumor, the size of a hen's egg, had developed at the hairy margin of the forehead." This contained glandular tubes, and stagnating secretion "led in many places to the formation of cysts ¼ to 5 mm. in size. . . . The tumor had no connection with the appendages over it, in particular, the sebaceous and sweat glands." Unna recorded a third case, described by Knause, of a tumor on the outer border of the foot of an old woman. A fourth case, that of Chandelux, was described as a "tuberculum dolorosum." It was of six years' duration and occurred near the elbow of a 43 year old woman, "a subcutaneous nodule, which was liable to spontaneous attacks of pain, and was exquisitely tender on pressure." It was the size of a pea, and consisted of

> A very dense, closely packed collection of epithelial cords, which were rolled and wound round each other. . . . The epithelial cords were covered by a thin membrane, and filled with very regular, polyhedric, angular epithelia. Only those directly seated on the membrane had a more cylindric form, and stained deeply in carmine, while the central cells, in general, were little stained and contained hardly visible nuclei.

Unna said that the Hoggans described a case similar to Chandelux's, and Unna also accepted the case of Audry and Nové-Josseraud as one

20. White, C. J.: Syringocystoma, J. Cutan. Dis. **25**:42 (Feb.) 1907 (including a voluminous bibliography).

of spiradenoma. He added one case of his own, which showed "a purely subcutaneous tumor, the size of a chestnut. . . . The coil glands immediately above the tumor are enclosed in abnormally dense connective tissue, and thus unrolled." Histologically he found a transition of the persistent coil glands into the tumor "by means of very fine epithelial cylinders," and in the areas composing the tumor, a "basis of fine cylinders . . . which show the type of coil glands, though in altered arrangement."

It is a little difficult, admittedly, to decide whether Unna saw what I observed in the case reported here. However his cases might be interpreted if one had first-hand observations of them, it is clear that Unna recognized a group of nodular tumors of sweat gland structure, and the case reported here is one of these.

7. The linear nevi made up of sweat glands or sebaceous glands, the seventh group of Paul and Adamson, are of a different order of abnormality. Unna recorded one such instance.[21] Rather than adenomatous activity of discrete anlagen, they are anomalies involving, in a circumscribed area, an abnormal quantity of relatively unaltered accessory structures.

SUMMARY

An unusual case presenting discrete nodules occurring unilaterally on the neck of a woman of 45 years is recorded. Histologic examination led to a diagnosis of a rare form of tumor of the sweat glands. The origin in the sweat glands was postulated on the observations that the tumor was of two-layered epithelial cylinder structure, and that less highly evolved nodules of syringoma occur at the periphery of larger nodules. The basic architectonic scheme of the nodules in this case is the secretory tubule surrounded by neoplastic proliferation of sheath cells. It is conjectured that a sudoriparous neoplasm with the growth urge emphasized on the secretory cell layer produces the classic syringocystadenoma, while if sheath cells are predominately active, then firmer, larger nodular growths occur, as seen in this case and in that of Paul and Adamson. The case described is an instance of tumor of the sweat glands, microscopically cystic and alveolar in structure, and clinically nodular in appearance; it is properly named syringocystadenoma nodularis.

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21. Unna,[11] p. 912.

CUTANEOUS DIPHTHERIA IN CONGENITAL SYPHILIS

REPORT OF A CASE

MILTON H. COHEN, M.D.
YORK, PA.

The following report describes a rather infrequent cutaneous complication in a patient suffering from congenital syphilis. Cutaneous diphtheria is a rare but not entirely uncommon manifestation, and congenital syphilis is found only too frequently in every hospital and clinic, but their occurrence in the same patient is unusual. In a search of the literature I was unable to find the description of a similar case. It is known that both Spirochaeta pallida and the Klebs-Löffler bacillus have a special affinity for the nasal passages, and it is interesting to note what happens when these two destructive forces invade the same nose.

REPORT OF CASE

History.—In January 1933, a girl, aged 11 years, came to the York Hospital Dispensary with an obvious chief complaint. Her nose was swollen and reddened, and a marked, odoriferous, mucopurulent discharge was present. Surrounding the alae for about 1 inch (2.5 cm.) was a crusted, raised patch resembling ecthyma except for the presence of a faint, gray veil which covered the lesion. On removing the crusts a raw, bleeding surface was revealed. The nostrils were filled with a foul exudate, and when this was removed the nasal septum was found to be completely absent. The cartilages, the subcutaneous tissues and the overlying skin had been destroyed and the nose had been kept from collapsing only by the presence of the thick, crusted discharge. The patient gave the date of onset of her trouble as May 1931, when she suffered severe attacks of epistaxis following trauma to her nose. Bleeding continued at intervals, and she gradually began to notice a foul odor and an increased nasal discharge. Soon the skin around the alae became red and swollen, and the lesion became progressively more severe.

Examination.—With the exception of the presenting symptom the physical examination revealed nothing abnormal. The heart and lungs were normal. The forehead was high, the base of the head broad and the skin sallow and pasty, and the general facial picture suggested heredosyphilis. The patient did not have pegged teeth, keratitis, nerve deafness, saber shins, nodes on the clavicles or other signs generally associated with congenital syphilis; nevertheless the Wassermann reaction was four plus. It was later discovered that her father was being treated for tertiary syphilis at another clinic. A roentgenogram of the skull revealed no evidence of necrosis of bone showing that all the damage was restricted to the cartilages and the soft parts of the nose. A urinalysis gave normal results, and a blood count showed: hemoglobin content, 80 per cent; red cells, 5,200,000, and white cells, 8,200.

Course and Treatment.—In view of the necrosis of the septum and the positive Wassermann reaction it was assumed that the cause of the ailment was syphilis, although the clinical appearance of the cutaneous lesion was considered strange, not resembling any known syphilid. She was given 0.45 Gm. of neoarsphenamine every five days for six doses and a 20 per cent solution of mild silver protein was applied locally. No change was noted in the appearance of the lesion, and the discharge and foul odor continued.

A culture was then made from the patch on the skin and also from the throat. A pure growth of Klebs-Löffler bacilli was obtained from both sites. It was later found that in the patient's immediate neighborhood five cases of diphtheria of a very malignant type had appeared, resulting in two deaths. She was given 10,000 units of diphtheria antitoxin, and in one week the nasal discharge had stopped, the crusts and membrane had loosened, and healing had begun to take place. After ten injections of neoarsphenamine were given a course of bismuth injections was started. About three weeks after the administration of the diphtheria antitoxin complete healing of all acute lesions had taken place. The nose collapsed after the supporting secretions were permanently removed, producing an

Left, patch of cutaneous diphtheria beneath the nose, with the absence of the nasal septum; right, results of treatment with diphtheria antitoxin and neoarsphenamine.

unsightly deformity, on which a plastic operation will be done after efforts are made to make the Wassermann reaction negative.

COMMENT

This case is instructive for more than one reason. First, it shows that a positive Wassermann reaction does not always explain every symptom a patient may have, and, second, the clinical doubt should have led to an immediate culture. Freidell,[1] in describing the difference between syphilis of the skin and cutaneous diphtheria, pointed out that in syphilis no membrane is present, there is no tendency to the formation of confluent areas, and the lesions are darker and more sluggishly inflammatory. These differences were well exemplified in this case.

It is interesting to contemplate the origin of the condition. Did the blow on the child's nose "light up" a latent diphtheritic condition? And

1. Freidell, A.: Skin Diphtheria, Minnesota Med. **8**:671 (Nov.) 1925.

what bearing did the underlying syphilis have on the production of the pathologic changes? The predilection of Spirochaeta pallida for the nasal cartilages must have had some part in producing the finished picture, but it must be remembered that the Klebs-Löffler bacillus also has a selective affinity for the nasal passages. According to Biberstein [2] every epithelial lesion permits the invasion of the diphtheritic bacillus. In large, deep lesions the infection is generally called surgical, or wound, diphtheria; in slight lesions, cutaneous diphtheria. He stated that a special allergic condition of the skin seems to be the prerequisite for the occurrence of cutaneous diphtheria. Since the intact skin is impermeable to the Klebs-Löffler bacilli it must be assumed in this case that the syphilitic condition altered the skin, either indirectly through an allergic manifestation or directly through pathologic changes in the tissue. In this connection it is interesting to note that Löhe and Rosenfeld [3] reported a case in which a positive Wassermann reaction was called forth by the injection of diphtheria serum in a nonsyphilitic patient. The positive reaction remained for three months and then disappeared spontaneously. However, the opposite result was found by Boas and Többöll [4] who performed Wassermann and Kahn tests on one hundred patients with diphtheria before and after serum treatment. A positive reaction was obtained in only one instance, and cleared entirely six days later. The whole question of allergy and the effect the presence of one disease may have on another is still an unsettled problem. In this case it is not possible to state definitely that the syphilitic rhinitis or the nasal diphtheria would have occurred independently, or what the result would have been if only one organism had been involved.

Although cutaneous diphtheria is rare, its presence should always be considered, particularly since it is a dangerous source of infection. Massey and Russel [5] reported a severe outbreak of diphtheria arising in a hospital from a nurse who had a patch of intertrigo behind the ear. This was later shown to be cutaneous diphtheria. Reinhardt [6] claimed that any ulcer or lesion resembling ecthyma, impetigo, intertrigo or pemphigus that is slow to heal, has tough borders and is covered with

2. Biberstein, Hans: Cutaneous Diphtheria, Klin. Wchnschr. **6**:1242 (June 25) 1927.

3. Löhe, H., and Rosenfeld, H.: Injection of Diphtheria Serum Causing a Positive Reaction in a Nonsyphilitic Person, Dermat. Ztschr. **53**:373, 1932.

4. Boas, Harold, and Többöll, Gudrum: Can an Injection of Diphtheria Serum Call Forth a Positive Reaction in a Nonsyphilitic Person? Dermat. Wchnschr. **94**:173 (Jan. 30) 1932.

5. Massey, Arthur, and Russel, Gladys J. C.: A Case of Cutaneous Diphtheria as the Cause of an Outbreak, British M. J. **1**:992 (June 1) 1929.

6. Reinhardt, Ilse: Cutaneous Diphtheria in Early Childhood, München. med. Wchnschr. **71**:610 (May 9) 1924.

a fine membrane should be examined for Klebs-Löffler bacilli. The lesion in this case, acting as a carrier source for many months, was the probable cause of several other cases of a particularly virulent type of diphtheria in the patient's community.

The diagnosis of cutaneous diphtheria is not always simple. Reinhardt [6] assumed that the presence in a lesion of an organism morphologically resembling the Klebs-Löffler bacillus and a favorable response to diphtheria antitoxin are sufficient criteria to establish a diagnosis. However, Edelstein [7] reported a number of cases in which Klebs-Löffler bacilli were found in various cutaneous lesions not suggesting clinical cutaneous diphtheria at all. It appeared that the bacilli were in symbiosis with streptococci. Knowles and Frescoln [8] emphasized the difficulty of differentiating the Klebs-Löffler bacillus from other organisms, and particularly warned against its confusion with the pseudodiphtheritic, or Hoffmann, bacillus. In my case the lesion showed a typical culture of Klebs-Löffler bacilli, and the lesion morphologically resembled the ecthyma type of cutaneous diphtheria. The presence of a false membrane, although this is not so essential to the diagnosis as the older authors assumed, the quick response to diphtheria antitoxin and the history of contagion in the neighborhood all served to establish the diagnosis.

SUMMARY

A case of cutaneous diphtheria was reported in a patient afflicted with congenital syphilis. The nose and surrounding tissues were chiefly affected, and the resulting nasal diphtheria and syphilitic rhinitis caused the complete destruction of the nasal septum, including its cartilages, the subcutaneous tissue and the overlying skin. Diphtheria antitoxin and antisyphilitic remedies alleviated the acute condition, but left a marked cosmetic defect.

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7. Edelstein, Halpert A.: Diphtheriebazillen auf der Haut von Säuglingen, Ztschr. f. Kinderh. **36**:84, 1923.

8. Knowles, F. C., and Frescoln, L. D.: Diphtheria of the Skin of Unusual Types; Report of Two Cases, J. A. M. A. **63**:398 (Aug. 1) 1914.

THE NEUROCUTANEOUS SYNDROME

THE CONGENITAL ECTODERMOSES

GEORGE V. KULCHAR, M.D.
SAN FRANCISCO
AND
LEONARD E. ANDERSON, M.D.
PHILADELPHIA

The often bizarre neurologic symptoms and signs occurring in conjunction with nevoid changes in the skin are perhaps more familiar to the neurologist than to the dermatologist. In most cases, however, the recognition of the nature of the cutaneous changes gives the key to the diagnosis. Since the skin and the nervous system have a common embryonic origin, namely, the ectoderm, aberrations in the genesis of this embryonal layer often result in congenital malformations affecting both structures selectively. To this varied group of neurologic signs and symptoms, following no true pattern and occurring in association with cutaneous nevi, the term neurocutaneous syndrome has been applied. The wide variety of neural and cutaneous pictures included in this syndrome is to be expected, inasmuch as the nature and degree of aberration from the normal will depend on the stage of embryologic development and differentiation reached by the embryonic ectodermal layer when the malforming factor becomes operative.

An attempt has been made by Yakovlev and Guthrie [1] to classify the heterogeneities included in this syndrome into three main groups, beyond which any orderly division seems impossible. Under the classification of these authors, the neurocutaneous syndrome, or the "congenital ectodermoses," as they term them, may be divided into (1) Recklinghausen's neurofibromatosis, (2) Bourneville's tuberous sclerosis and (3) the encephalotrigeminal vascular syndrome (angiomatosis of the brain associated with cutaneous nevi in the area of trigeminal distribution).

Multiple neurofibromatosis has long been familiar to dermatologists since the classic description by von Recklinghausen [2] in 1882. The

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From the Department of Dermatology and Syphilology, School of Medicine, University of Pennsylvania, Philadelphia, John H. Stokes, M.D., Director.

1. Yakovlev, P. I., and Guthrie, R. H.: Congenital Ectodermoses (Neurocutaneous Syndromes) in Epileptic Patients, Arch. Neurol. & Psychiat. **26**:1145 (Dec.) 1931.

2. von Recklinghausen, F.: Ueber die multiplen Fibrome der Haut und ihre Beziehung zu den multiplen Neuromen, Berlin, A. Hirschwald, 1882, p. 138.

fibromatous tumors occurring in the skin and along the peripheral nerve trunks and the concomitant presence of *café au lait* spots are seen too often to merit more than passing mention. The association of multiple neurofibromatosis with lowered mentality has long been noted, although it is likewise seen in patients of apparently normal mentality. Less constant is the presence of the nevus anaemicus of Vörner and the familiar vascular or "port wine" nevus in patients with Recklinghausen's neurofibromatosis. Both the nevus anaemicus of Vörner and the "port wine" nevus are considered by Weber [3] as being neurogenic in nature and secondary to a congenital defect in the vasomotor innervation causing a permanent vasospasm in the former and a permanent vasodilatation in the latter.

The tuberous sclerosis of Bourneville is a more clearly defined syndrome, and its association with epilepsy and mental defect is most constant, according to Yakovlev and Guthrie. Clinically the dermatologist is familiar with the cutaneous phase of this syndrome under the term of adenoma sebaceum. In addition, however, to the occurrence of adenomatous papules and nodules over the cheeks and forehead and occasionally elsewhere on the body, mental defect or epilepsy is present in many instances. The association of other nevoid changes such as angiomas and von Recklinghausen's neurofibromatosis with adenoma sebaceum has long been noted. Jadassohn [4] has shown that the lesions of adenoma sebaceum are not due to an adenomatous or a hyperplastic state of the sebaceous glands, but are nevoid in character, being made up of hyperplastic connective or vascular tissues. In addition to the nevoid cutaneous changes described in connection with Bourneville's tuberous sclerosis, multiple firm nodules distributed over the surface of the brain with a predilection for the cortex of the frontal lobes are found at necropsy. Bielschowsky and Freund [5] have shown that these nodules are the result of an abnormal proliferation of the undifferentiated spongioblasts or, in other words, a primitive gliosis. Likewise in the internal viscera (kidneys, heart, pancreas, liver and ovaries) congenital anomalies such as tumors and cysts are sometimes noted in cases of tuberous sclerosis.

More recently, considerable attention has been directed to the association of angiomatosis of the brain with cutaneous nevi distributed over

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3. Weber, F. P.: The Relation of Capillary Hemangiectatic Naevus and Naevus Anaemicus to the Nervous System, Brit. J. Dermat. **41**:221 (June) 1929.

4. Jadassohn, J.: Die benignen Epitheliome, Arch. f. Dermat. u. Syph. **117**: 577, 704 and 833, 1914.

5. Bielschowsky, M., and Freund, C. S.: Ueber Veränderungen des Striatums bei tuberöser Sklerose und deren Beziehungen bei anderen Erkrankungen dieser Hirnteils, J. f. Psychol. u. Neurol. **24**:20, 1918.

the trigeminal area of the face. Cushing and Bailey,[6] among others, have pointed out the frequent association of venous angiomas in the brain with vascular nevi in the distribution of the trigeminal nerve. The association of cerebellar cysts arising from hemangiomas with an angiomatosis of the retina (Hippel's disease) has been noted by Lindau.[7] The angiomatoses of the central nervous system occurring in association with similar vascular anomalies in other structures of ectodermal origin have been divided into two types by Lévy.[8] To the association of cerebellar vascular anomalies with an angiomatosis of the retina (Lindau-Hippel's disease) he has given the name of neuro-ocular syndrome. The association of cutaneous vascular nevi with an angiomatosis of the corresponding neuromeres of the central nervous system is classified by Lévy as being representative of the neurocutaneous syndrome.

Since the blood vessels are of mesodermal origin, the inclusion of vascular anomalies in the group of congenital ectodermoses may be questioned. Yakovlev and Guthrie, from a clinicopathologic consideration, stated the opinion that the hyperplasia of the mesodermal tissues (blood vessels) is superimposed on the background of the congenital malformation of the neuroectodermal tissues. According to these authors:

> The congenital ectodermal defect reflecting itself electively on the vasomotor innervation may be looked on as at least one of the factors in the capillary, venous and arteriovenous telangiectatic hyperplasia in the corresponding segments of the skin and in the central nervous system.

On this ground they considered that angiomatoses of the central nervous system in association with cutaneous vascular nevi are an ectodermal malformation of the vascular type, as viewed from the pathogenic rather than the histiogenic standpoint. Roussy and Oberling[9] lent further support to this conception by advancing the view that in the congenital anomalies which appear to be of mesodermal origin there is also an ectodermal element, namely, a gliosis. The gliosis assumes an active rôle in the congenital anomaly as a primitive glial process in combination with vascular hyperplasia or even formation of tumor.

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6. Cushing, H., and Bailey, P.: Tumors Arising from the Blood Vessels of the Brain: Angiomatous Malformations and Hemangioblastomas, Springfield, Ill., Charles C. Thomas, Publisher, 1928, p. 211.

7. Lindau, A.: Studien über Kleinhirncysten; Bau, Pathogenese und Beziehungen zur Angiomatosis retinae, Acta path. et microbiol. Scandinav. (supp. 1), 1926, p. 1.

8. Lévy, G.: L'angiomatose du système nerveux central, Presse méd. **38**:37 (Jan. 8) 1930.

9. Roussy, G., and Oberling, C.: Les tumeurs angiomateuses des centres nerveux, Presse méd. **38**:179 (Feb. 5) 1930.

The following case is an example of the neurocutaneous syndrome, which displays the most unusual combination of adenoma sebaceum-like lesions over the face with mental deficiency, in addition to extensive fibrovascular nevi over the extremities and evidence of anomalies of the spinal cord.

REPORT OF A CASE

History.—L. S., a white man, aged 29, was first seen in the Clinic of Cutaneous Medicine and Syphilology on Dec. 8, 1933, complaining of a deep-seated, dull pain over the buttocks and lower extremities for the past nine years, which was increased by exertion. There had been an increasing spasticity of the lower extremities with progressive difficulty in walking for the past four years. Inquiry

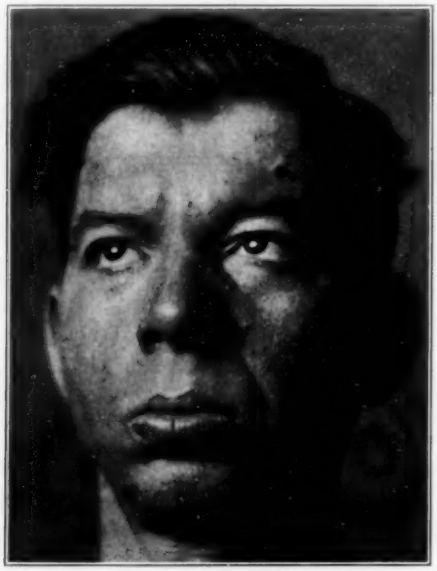

Fig. 1.—Multiple nevi of the face.

into the family history revealed that ten siblings had died in early childhood, one supposedly of a spina bifida and the remainder of unknown causes. So far as the history was obtainable, none of the other members of the family had noted nevi or were afflicted with a neurologic disorder. There was no history of consanguinity in marriage. The past medical history of the patient was essentially of no consequence, and there had been no "spells" or epileptic seizures.

Physical examination revealed an underdeveloped man, somewhat mentally retarded, who walked with an obvious spastic gait. The head was moderately scaphocephalic, but no ridge along the sagittal sutures could be made out. There was an exostosis about 2 cm. in diameter over the occiput. The hair and teeth were normal in appearance. The hands were disproportionately large, with bilateral dactylomegaly of the fourth and fifth fingers. The feet, however, were normal except for syndactylism of the fourth and fifth toes bilaterally.

There was a rosaceiform erythema of the entire face, most marked in the central oval. Scattered over the cheeks and forehead and particularly about the

nasolabial groove were a number of erythematous papules and nodules, varying in size from 3 mm. to 7 mm. in diameter. With a hand lens, minute telangiectatic capillaries could be seen coursing over the surface of the lesions. In a strikingly symmetrical distribution over the forearms, antecubital region, hands and lower extremities, extending well above the midthigh, there were sharply marginated, infiltrated, confluent nevi, which the patient stated had been present since birth without any evident change. The nevi were deeply erythematous in color and raised; they involved the entire forearm and legs in a confluent "glove" or "stocking" distribution except for symmetrical islands of skin of normal appearance on

Fig. 2.—Multiple nevi of the extremities and skeletal anomalies.

the legs. Over the hands and the lower part of the forearms the nevi appeared abnormally dry and hyperkeratotic, with marked accentuation of the normal furrows of the skin.

Neurologic Examination.—Mentally the patient was below normal, and his general information, concentration, attention and judgment were impaired, although there was no profound mental defect. There was mild general muscular weakness, more marked on the right side and relatively greater in the lower extremities. The gait was spastic, the right lower extremity being more spastic than the left. All of the tendon reflexes were greatly exaggerated, but equal. A pronounced jaw jerk and a bilateral Trömmer reflex were noted. Ankle and patellar clonus which was most marked on the right side was elicited. The Babinski, Gordon and

Oppenheim reflexes were negative. Because of the defect of attention it was impossible to judge the accuracy of milder degrees of sensations. However, there was no gross impairment of pain, touch, temperature and vibratory sensations. At times fascicular and wavelike contractions were noted in the shoulder girdle and in the gluteal muscle groups.

There was a suggestion of pallor of the optic disks, but no retinal changes or angiomas could be detected on ophthalmoscopic examination.

Laboratory Examination.—The blood count was normal, and urinalysis gave essentially normal results. The Wassermann (Kolmer and Kahn) tests of the blood were negative. A lumbar puncture revealed no evidence of a spinal fluid block and the pressure was normal. An examination of the spinal fluid gave negative results, except for an increase in protein (15 units). Roentgenograms of the skull and long bones showed nothing abnormal. Roentgen studies of the vertebrae showed lumbar scoliosis.

Biopsy.—A section was taken from the left thigh. Dr. Weidman's report is as follows: "The epiderm showed a high grade of acanthosis, being thickened up to five or six times the normal and exhibiting closely placed, broadened and more or less fused interpapillary pegs. There was a moderate thickening of the stratum corneum in this connection. In the corium the only abnormalities discoverable consisted in slight hyperplasia of the capillaries, which nevertheless maintained a fairly normal caliber. There were no inflammatory features. The sweat and sebaceous glands were not represented. The hair follicles were definitely enlarged, and the hair shafts were correspondingly heavier.

"The only definite evidence of nevoid change consisted in the exaggerated size of the hair follicles. The minor hyperplasia and dilatation of the capillaries in the papillae and the overlying acanthosis could just as well have been due to a long-continued, low grade irritation. In the light of the clinical data it must be assumed that the congenital vice was affecting the epiderm and inducing an order of change such as is seen in linear nevus, but of lesser degree."

Dr. M. J. Cooper, of the department of neurology, saw the patient in consultation and rendered the following opinion: "The picture points toward a diffuse degenerative process in the cord, bilaterally, and might best be accounted for on the basis of an abiotrophy affecting the pyramidal tracts primarily with damage to the anterior horn cells or a gliosis affecting the same tracts and cells. The other congenital anomalies and the history of the early death of ten siblings, one probably as a result of spina bifida, lend support to the conception of a congenital system defect or a predisposition to gliosis as the basis of the neurologic condition."

COMMENT

The case reported does not conform to any of the three heretofore recognized types of the neurocutaneous syndrome. The widespread distribution of the congenital anomalies involving the face, extremities, spinal cord and probably the brain is the most unusual feature. The occurrence of the nevi on the extremities in association with changes in the central nervous system is infrequent in the heretofore reported cases of the neurocutaneous syndrome. Cobb [10] reported a somewhat similar case in which the nevi were few and small, while the lesion of the

10. Cobb, S.: Haemangioma of the Spinal Cord Associated with Skin Naevi of the Same Metamere, Ann. Surg. **62**:641 (Dec.) 1915.

spinal cord was localizable at the level of the same metamere. In the case reported here it must be assumed, in view of the wide distribution of the nevi and the signs indicating an extensive abiotrophy or a gliosis of the central nervous system, that the malforming factor must have become operative before the ectoblast had reached any considerable degree of differentiation.

Skeletal anomalies such as are presented by this patient were reported by Goodhart [11] as occurring in Recklinghausen's neurofibromatosis. Brooks and Lehman [12] cited scoliosis as being among the characteristic changes in the bone in Recklinghausen's disease. Whether the scaphocephaly, dactylomegaly, syndactylism and scoliosis noted in the patient reported here are analogous changes must remain conjectural, but it is not beyond the realm of possibility that a malforming factor becoming operative early in the stage of embryogenesis might result in skeletal anomalies.

The possibility that lowered mentality in the patient may be the result of Bourneville's tuberous sclerosis involving the cortex of the frontal lobe must await confirmation at necropsy. The association of adenoma sebaceum with tuberous sclerosis as one type of the neurocutaneous syndrome according to the classification of Yakovlev and Guthrie has been cited earlier.

SUMMARY

Since the skin and the central nervous system arise from a common source, the ectoblast, a wide variety of congenital anomalies affecting these two structures selectively may be found in association.

The nature and the degree of aberration from normal in the congenital anomalies observed depend on the stage of embryologic development and differentiation reached before the malforming factor becomes operative.

Reported cases have been classified into three main groups: (1) Recklinghausen's neurofibromatosis, (2) Bourneville's tuberous sclerosis and (3) the encephalotrigeminal vascular syndrome. However, many variants and transitional forms are observed.

A case presenting extensive multiple nevi of the face and extremities, with widespread involvement of the spinal cord and probably of the brain and skeletal anomalies, is reported as a variant of the neurocutaneous syndrome.

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11. Goodhart, P.: Neurofibromatosis, in Tice, F.: Practice of Medicine, Hagerstown, Md., W. F. Prior Company, Inc., 1927, vol. 10, p. 665.

12. Brooks, B., and Lehman, E. P.: Bone Changes in Recklinghausen's Neurofibromatosis, Surg., Gynec. & Obst. **38**:587 (May) 1924.

PACHYONYCHIA CONGENITA JADASSOHN

A VARIETY OF ICHTHYOSIS (PACHYONYCHIA ICHTHYOSIFORMIS) INVOLVING CHIEFLY THE NAILS

F. A. DIASIO, M.D.
Dermatologist and Syphilologist, Columbus and Saint Elizabeth's Hospitals, and Associate Dermatologist, Lincoln Hospital
NEW YORK

Under the caption "Congenital Dyskeratosis," Erich Schäfer [1] described a syndrome based on observations gathered from one case of his own and from thirteen others reported in the literature. The condition is characterized by: pachyonychia; acneform, follicular keratosis, especially about the knees and elbows; symmetrical and circumscribed plantar and palmar keratosis; hyperhidrosis of the palms and soles; leukoplakia of the oral mucosa, and anomalies of the hair. All the fourteen cases showed pachyonychial changes as a constant finding, while the remaining clinical features were variants.

In Schäfer's case a boy, 12 years old, had had the characteristic ungual manifestations from birth. Brettauer's report [2] dealt with a boy 9 years old. The finger-nails were thickened, opaque, lusterless, partially loosened and in places swollen and lap-rolled in the longitudinal axis of the finger. According to Heller,[1c] Jadassohn and Lewandowsky's case [3] of pachyonychia congenita belongs in this group. Siemens [4] likewise reported an example of congenital dyskeratosis under the title "Keratosis Follicularis." Babicèk [5] recorded an instance of plantar and palmar keratosis which was neither inherited nor familial. Andrews and Strumwasser's patient [6] began to show pachyonychial changes two weeks after birth. Fox's patient [7] also presented thickening of the nails and other suggestive features of this disorder.

From the Department of Dermatology and Syphilology, Columbus Hospital.

1. (*a*) Schäfer, E.: Arch. f. Dermat. u. Syph. **748**:425, 1924. (*b*) Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1927, vol. 13, pt. 2, p. 86. (*c*) Heller, Julius: Die Krankheiten der Nägel, ibid., p. 87.

2. Brettauer, cited by Heller.[1c]

3. Jadassohn, J., and Lewandowsky, in Neisser, A., and Jacobi, E.: Ikonographia dermatologica, Berlin, Urban & Schwarzenburg, cited by Heller.[1c]

4. Siemens: Arch. f. Dermat. u. Syph. **139**:62, 1922.

5. Babicèk: Rev. de med. Tchèque **1**:5, 1920; cited by Heller.[1c]

6. Andrews and Strumwasser: New York State J. Med. **29**:747, 1929.

7. Fox, H., cited by Andrews and Strumwasser.[6]

Associated defects have been described. In the patients seen by Bettmann,[8] Jadassohn and Lewandowsky,[3] Strandberg [9] and Schäfer [1] bullae appeared from time to time on the thickest parts of the soles. Andrews and Strumwasser's [6] patient had herpetic lesions on the oral mucosa at intervals. In the majority of cases keratosis of the mucous membranes of the mouth (leukoplakia) has been noted. In Burns' [10] patient, a boy, the nasal mucosa was similarly affected while the tympanic membranes were so thickened and retracted that the boy was totally deaf.

Opacities and partial blindness due to dyskeratosis of the cornea have been reported by Brettauer,[2] Brunauer [11] and Burns.[10] Riehl's patient,[12] a Bulgarian, had abnormally long bones, excessive motility of the joints and the striped or linear form of palmar keratosis. The boy seen by Schäfer [1] had bilateral cataract and twinning of the incisor teeth. Mental deficiency was noted by Brunauer.[11]

Pachyonychia congenita of Jadassohn affects chiefly males and appears to be most prevalent among Slavs and Jews of Slavonic origin. It is rarely represented in the sibships of twins, the notable exception being the boy seen by Mendes da Costa and van der Valk,[13] who was one of triplets.

REPORT OF A CASE

The patient was a school boy, 9 years old. Notwithstanding his Italian parentage he had light blue eyes, a fair complexion and light brown hair. He resembled very much the male parent, who had been treated for syphilis about twenty years previously. Several years before he married, the Wassermann reaction of the blood had been reported negative. The mother's blood had given a negative Wassermann reaction before her only child was born. There was no consanguinity of the parents, nor had any abnormality similar to that displayed by the patient been reported in the grandparents, ascendants or collaterals. The patient had been normal at birth. His infancy and childhood were uneventful except for an attack of measles, which was associated with respiratory complications.

Four years before I saw the patient the periungual tissues of the middle finger of the left hand became red and boggy. A physician removed the nail plate. Six months later the patient injured the great toe of his left foot. Subsequently all the nails of the left foot became red and boggy. Another physician extracted the diseased nails. Within a few weeks the toe-nails of the right foot became involved, for which roentgen treatments were given, with aggravation of the condition. The mother then proceeded to take the matter in her hands and pulled out all the loosened nails.

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8. Bettmann, in Schwalbe, Ernst: Die Morphologie der Missbildungen des Menschen und der Tiere, Jena, Gustav Fischer, 1909, p. 735.

9. Strandberg, cited by Cockayne, E. A.: Inherited Abnormalities of the Skin and Its Appendages, New York, Oxford University Press, 1933, p. 206.

10. Burns, cited by Cockayne.[9]

11. Brunauer: Dermat. Ztschr. **42**:6, 1924.

12. Riehl: Zentralbl. f. Haut- u. Geschlechtskr. **6**:335, 1922.

13. Mendes da Costa and van der Valk: Urol. & Cutan. Rev. **23**:159, 1919.

Other important features in the history were: (1) hyperhidrosis confined to the palms and soles, of several years' duration; (2) dry, itchy skin and marked pityriasic scaliness of the scalp since the age of 2 years; (3) epiphora (right eye) for two years; (4) hemangioma at the tip of the tongue, first noticed soon after birth (the mother stated that the vascular tumor would from time to time enlarge and simulate a strawberry), and (5) slight deafness.

The patient was examined in a clinic for cutaneous diseases. On the tentative diagnosis of syphilitic onychia, he received a course of eleven intramusclar injections of a bismuth compound and eight intravenous injections of neoarsphenamine, without clinical improvement. Three Wassermann tests of the blood were negative, and repeated mycologic examinations of the nails were uniformly negative. There was also no response to local fungistatic therapy.

My examination revealed the boy to be neither mentally deficient nor physically inadequate. There was no evidence of hypogenitalism; however, the testicles were only partially descended. The skin was xerotic throughout. A fine pityriasic scaliness was noted on the scalp, face and trunk. On the cheeks, ears and margins of both eyelids the skin was so thin that the underlying vascular network was visible (telangiectatic), and over the extensor surfaces of the extremities it was studded by many keratotic follicles (lichen pilaris).

The hair of the scalp was dry and lusterless. There were no corneal opacities or cataracts present. The conjunctivae were clear. The lacrimal ducts of the right eye were not patent, for the ophthalmologist was unable to pass a probe in spite of repeated attempts. The obstruction of the ducts, accounting for the unilateral epiphora, was probably due to the same ichthyotic process noted elsewhere. The tympanic membranes were slightly thickened and retracted. There were no dental defects, such as twinning of the upper incisors. The anterior two thirds of the tongue was splashed by irregular patches of a smooth, white infiltration (leukoplakia, fig. 1). There was neither smooth atrophy of the tongue nor interstitial glossitis. On the tip of the tongue a reddish, smooth, rounded, nut-sized nodule, which could be partially dehematized, was visible. There was no abnormality of the thyroid gland.

On the elbows were patches composed of grouped pinkish, pea-sized papules which were capped by fine, adherent scales. Similar patches were noted over both knees, except that on the left kneecap the papules were discrete and distributed in an irregular fashion. Many discrete pinkish papules, intermingled with typical lesions of lichen pilaris, were scattered over the extensor surfaces of all the extremities.

The skin of the palms was glazed, moist and slightly thickened, but was not as keratotic as in keratoderma palmaris; the surface of the soles was likewise hyperhidrotic and somewhat cornified. The skin over the terminal phalanges was dry, erythematous and thinned, while the tips of the fingers and toes suggested "clubbing" (fig. 2).

The finger-nails presented a bizarre picture. Some were thickened, yellowish white, loosened laterally (over the distal third of the nail bed) and lap-rolled to describe the letter O when viewed anteriorly. Other nails were absent, so that the concave nail bed was covered by a dark, horny pellicle. The remainder were deformed (gryphotic), opaque and longitudinally furrowed, and displayed the stairstep-like exfoliation. The nails of the great toes were dark, unevenly thickened (due to subungual keratosis and heaping up of the nail plate) and shortened anteroposteriorly. The remaining toe-nails were thin (excessive exfoliation?), tapered in an upward direction and lacked the lunula, which was also absent on the finger-nails.

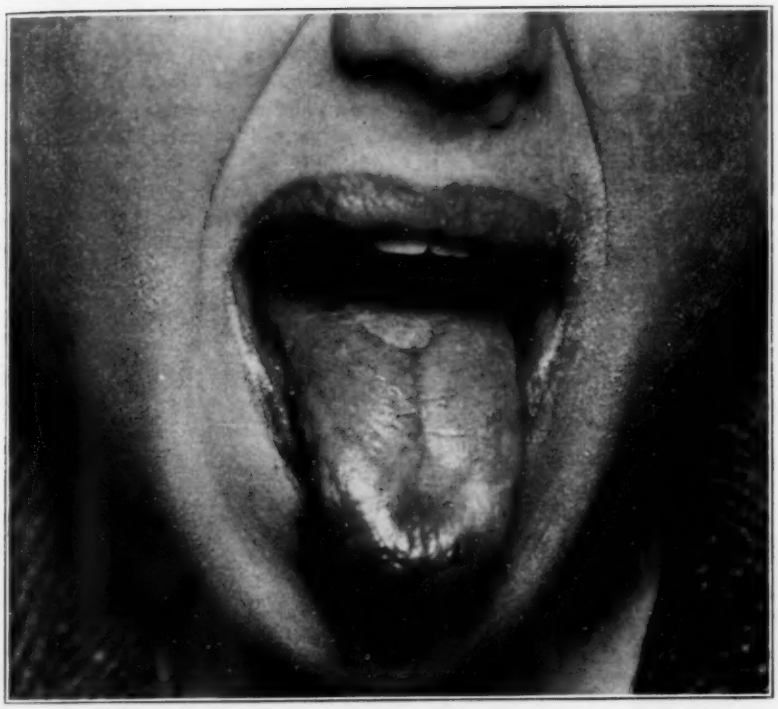

Fig. 1.—The smooth, white infiltration of the tongue and the scaliness of the skin are evident.

Fig. 2.—The condition of the finger-tips and the lap-rolling and thickening of the nails are notable.

While the patient was under observation it was noticed that the subungual keratosis would lift up the free margin of the nail which was about to be shed. The free margin would become thicker, darker and more opaque, while the nail plate loosened laterally. Eventually the plate would be shed, leaving a dark pellicle (fig. 2).

Routine laboratory investigations gave negative results. A biopsy specimen taken from the patch over the right knee was examined by Dr. D. L. Satenstein.

Fig. 3.—A section showing the increased horny cell layer with lamellation. The granular cell layer is thickened. The rete cells are small and show premature keratinization.

The most conspicuous features of the sections examined were (fig. 3): (1) a marked thickening of the horny layer, which was composed of loose, horny lamellae; (2) a relative thickening of the prickle cell layer, in which the cells were decreased in volume owing to premature keratinization; (3) a sparse cellular infiltration about the vessels in the upper cutis; (4) large size of the lumen of the sweat glands and small size of the sebaceous glands; (5) hypertrophy of the collagen bundles and no disappearance of the elastic tissue or fat; (6) cornification of the follicular openings, but not of the sweat pores, and (7) presence of the

granular layer throughout, though less marked in some areas than in others, and lack of nuclei in the horn cells.

A section of one of the nails revealed a swelling of the horn cells making up the nail plate. The horn cells retained their globular outline and presented evidence of degeneration (fig. 4).

The patient was given small doses of thyroid extract. The skin was anointed daily with a bland cream, and a weak sulphur ointment was suggested for the nails. The results have not been satisfactory.

Fig. 4.—Photomicrograph showing swollen horn cells and degenerative cells.

COMMENT

The formation of keratin is a specific function of the epithelial cells. Dyskeratosis is an underlying disturbance of this specific function, i. e., there is an interference with the aging process of epithelial cells. The abnormal cornification may be characterized by (1) faulty development or (2) abnormal behavior. The change is most marked in the horny layer. The dyskeratotic process (i. e., the disturbance in the evolution

of epithelial cells with special reference to cornification) may, according to Satenstein's classification,[14] be accelerated, resulting in a premature senility of epithelial cells, or it may be retarded, causing delayed juvenility of these cells. In the former one finds the dry, and in the latter the wet, type of dyskeratosis. In both types the basal cell layer may be normal.

The disturbed keratinization may affect isolated cells or groups of cells, sections, layers or even the entire epidermis. Moreover, it may or may not be accompanied by inflammatory manifestations in the cutis, epidermis or both. Finally, the dyskeratotic process may be the predominating change or simply a secondary feature.

Ichthyosis is an example of the dry type of dyskeratosis, for it is characterized by premature keratinization owing to premature aging of the prickle cells. This faulty or improper aging of the epithelial cells results in (1) a horny layer which may be normal or increased to a varying extent, depending on the rapidity of premature keratinization, producing loose, horny lamellae; (2) a granular layer which may be normal or increased owing to premature keratinization; (3) a thinned or thickened prickle cell layer showing keratinization as well as shrinking of the affected cells; (4) an increased number of mitoses in the basal cell zone, and (5) a papillomatous configuration of the epidermis without a verrucous surface. The horny layer progresses down the follicles and sweat pores. The follicular cornification is most marked on the extensor surfaces.

Gassmann's histologic investigations of ichthyosis and the ichthyotic-like diseases[15] are in agreement with the foregoing summary of the pathologic process in ichthyosis.

While ichthyosis has sites of predilection, the integument as a whole may be involved to a varying extent. The hyperkeratosis is commonly of varying degree and intensity. The involvement is usually unequal; thus, in the average case, the lesions on the extensor surfaces and on the lower extremities are more severe. On the other hand, the brunt of the dyskeratosis may sometimes be borne principally by structures such as the nails, which are infrequently involved. It then happens that the unusual variant is overlooked either because the changes occur in parts which are ordinarily not involved or because they are not well known.

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14. Satenstein, D. L.: Personal communications.

15. Gassmann, A.: Histologie und klinische Untersuchungen über Ichthyosis, Arch. f. Dermat. u. Syph. (supp.) **69**:5, 1904.

This failure to recognize aberrant types in the past has resulted in controversies (Unna,[16] Kaposi,[17] Neisser[18] and others) as to the existence of localized or circumscribed forms of ichthyosis in connection with mal de Meleda, keratoderma palmaris et plantaris, etc. Moreover, by naming differently the varied combinations of this disorder (ichthyosis) clinical entities, supposedly new, have been described while the nosologic status of the numerous variants has apparently been obscured.

An instance of this inaccurate classification is pachyonychia congenita of Jadassohn, of which my patient's condition is a typical example. The condition has been reported under various titles. It is characterized by the typical changes in the nails and the associated lesions of the skin and mucous membranes already alluded to. The histopathologic changes are those of the dry or accelerated type of dyskeratosis, which is featured by premature keratinization.

Since the dry type of dyskeratosis is the essential change in ichthyosis, and since the clinical features of pachyonychia congenita (Jadassohn) are typical of ichthyosis with the addition of the ungual changes, I believe that Jadassohn's pachyonychia is a rare variety of ichthyosis in which the nails show the most marked and most constant features.

The fact that pathologic nails are not frequently observed in ichthyosis, even in the recorded severe types, does not warrant the exclusion of pachyonychia congenita from the ichthyosis group of dermatoses. Moreover, the line of demarcation between ichthyosis and the several varieties of keratoderma[19] cannot be drawn, for in both premature keratinization is manifested by premature aging of the prickle cells.

According to Cockayne,[20] when sufficient data have been collected, pachyonychia congenita together with several other abnormalities of the skin will probably be found to be the result of an inheritance of a double dominant, in which (1) direct descent is rare in comparison with familial and sporadic incidence and (2) seldom continues for more than

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16. Unna: Ueber das Keratoma palmare et plantare hereditarium, Arch. f. Dermat. u. Syph. **10**:231, 1883; cited by Bohm: Dermat. Zentralbl., March 1904, p. 162.

17. Kaposi: Text Book of Skin Diseases, translated by J. C. Johnston, New York, William Wood & Company, 1895; cited by Bohm.[16]

18. Neisser, Lowenheim and Neumann, cited by Bohm.[16] von Zderas, O. H., and Ehlers, E.: Mal de Meleda, Arch. f. Dermat. u. Syph. (supp.) **40**:251, 1897. Sims, C. F.: Keratoderma Palmaris et Plantaris, Arch. Dermat. & Syph. **26**: 635 (Oct.) 1932.

19. Füellenbaum: Urol. & Cutan. Rev. **32**:277, 1928. Fuhs: Zentralbl. f. Haut- u. Geschlechtskr. **22**:316, 1927. Füellenbaum and Sawicka: ibid. **22**:627, 1927.

20. Cockayne, E. A.: Inherited Abnormalities of the Skin and Its Appendages, New York, Oxford University Press, 1933, p. 206.

two generations. These apparently explain the fact that in my patient the ectodermal defect appeared to arise *de novo*.

Since the term "congenital dyskeratosis" does not convey an exact impression as to what the outstanding clinical features of this disorder are and while Jadassohn's caption does not suggest the pathologic aspects, I propose the title "pachyonychia ichthyosiformis," which I believe meets all the requirements.

SUMMARY

1. A typical case of pachyonychia congenita Jadassohn in an Italian boy is recorded.

2. A survey of the literature and a study of the patient force the conclusion that Jadassohn's pachyonychia is a variant of ichthyosis affecting chiefly the nails.

3. The designation "pachyonychia ichthyosiformis" is suggested for the condition.

19 East Ninety-Eighth Street.

DERMATOMYOSITIS AND DIFFUSE SCLERODERMA

DIFFERENTIAL DIAGNOSIS AND REPORTS OF CASES

W. GEORGE BROCK, M.D.
Fellow in Dermatology and Syphilology, the Mayo Foundation
WINNIPEG, CANADA

This study was undertaken because of the apparent difficulty in distinguishing between dermatomyositis and scleroderma. A review of the entire literature on dermatomyositis was made to determine how distinctive a syndrome it presented. Not infrequently the difficulty of distinguishing scleroderma from dermatomyositis was mentioned, and in other instances it seemed that scleroderma could readily be considered in the diagnosis.

The following reports of cases illustrate the difficulties in differential diagnosis, and from them certain distinctive features can be pointed out.

REPORT OF CASES

CASE 1.—A housewife, aged 34, was the mother of two healthy children. Her family history and past illnesses were irrelevant. She had overworked for several years when, in April 1930, a red macular eruption appeared on the thorax, face, arms and hands. In four months weakness of the arms and legs supervened; this seemed to be aggravated following a tonsillectomy. In October, seven months after the onset of the condition, she became bedridden, was unable to move even her head, and had difficulty in swallowing. Her face and extremities became edematous and her muscles tender. Bronchopneumonia ensued. In February 1931 the edema faded, revealing atrophic muscles about the pelvic and shoulder girdles and the hands. After a short period of improvement, a relapse occurred.

Examination of the patient at the Mayo Clinic in September 1931 revealed signs of recent loss of weight. There were edema and mottled erythema of the face, neck, shoulders and thorax. The skin felt indurated and leathery in these regions. The muscles of the shoulder girdle, upper extremities and hips were hard, stringy, atrophic and weak. The lower extremities were relatively stronger. Movements were limited about the elbows, knees, shoulders and wrists. The knees and elbows presented contractures. The soft palate and the pharynx were weak. The skeletal muscles were tender. There were no changes in sensation. The temperature was normal. The blood pressure was 135 systolic and 70 diastolic; the pulse rate, 90. Except for these observations physical examination, urinalyses, blood cell counts, roentgenograms of the thorax, the Wassermann test and cultures taken from biopsy specimens of skin and muscle gave normal results. Six weeks

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Abridgment of a thesis submitted to the faculty of the Graduate School of the University of Minnesota in partial fulfilment of the requirements for the degree of Master of Science in Dermatology and Syphilology.

of hospitalization and treatment with nonspecific protein in graded doses (typhoid vaccine intravenously) produced some improvement. The patient went home and in two months suffered a severe relapse.

On admission to the University Hospital, Minneapolis, weakness was so general and severe that the patient could not feed herself. The slightest motion caused muscular pains. In all respects, this relapse was identical with the previous ones except that it was more severe. Because of the difficulty in swallowing, mucus collected in the throat; bronchopneumonia developed, and the patient died on March 3, 1932. The laboratory examinations had revealed nothing significant other than an almost pure culture of hemolytic staphylococci which had been obtained from the sputum. Permission for a postmortem examination was not obtained.

Biopsies of skin and muscle obtained while the patient was at the clinic in August 1931 revealed the following: The only essential changes in the skin were moderate edema of the papillaris, subpapillaris and cutis, and a proliferation of endothelial cells in the subpapillary vessels with a few young fibroblasts around the periphery. The arrectores pilorum muscles were swollen. No notable change had occurred in the subcutaneous tissues. The sections of muscle disclosed swelling of the individual fibers with indistinctness of cross-striations, separation of the longitudinal fibrils, hyaline and vacuolar degeneration, fragmentation and granular disintegration. There was a multiplication of nuclei in the muscle fibers. The vessels presented endothelial swelling and proliferation; in focal areas they were surrounded by dense mantles of lymphocytes, plasma cells and a few fibroblasts.

Case 2.—A girl, aged 12½ years, although previously healthy, had noted in March 1925 that her hands and feet turned blue, especially when dependent. Two months later all her muscles became sore, stiff, weak and weary. In July severe stomatitis and puffiness and erythema of the eyelids developed; this soon spread over the face, neck, arms and hands and later, over the thorax. The weakness and painfulness of the muscles became so severe that the patient was practically helpless. The pains were cramplike. Throughout the illness she suffered from diarrhea without pus or blood in the stool. She frequently complained of choking sensations.

On examination, Jan. 14, 1926, the patient appeared to be listless; edema and diffuse erythema of the eyelids, face, thorax, hands and knees were noted. The muscles were weak, tender and painful. The constrictor muscles of the hypopharynx appeared to be weak. The heart was slightly enlarged. The first sound was roughened over the mitral region. The blood pressure was 112 systolic and 60 diastolic; the pulse rate, 105, and the temperature, 99 F. The spleen was palpable three fingerbreaths below the costal margin. The patient was moderately anemic; the erythrocytes numbered 3,740,000 and the leukocytes 7,000 per cubic millimeter of blood; the hemoglobin was 60 per cent, and there were 8.5 per cent of eosinophils. Urinalyses, the Widal and Wassermann reactions, cultures of the blood and roentgenograms of the thorax and sinuses gave normal results. The stools were negative for pus, ova, parasites or blood. No trichinae were found on biopsy of muscular tissue. The basal metabolic rate was plus 17 per cent, the increased rate being probably the result of recent thyroid medication. From a culture taken from muscle Dr. Rosenow isolated a streptococcus that produced muscular lesions in rabbits and hemorrhages into the skin after intravenous injection. The patient died of bronchopneumonia four months later at her home, fourteen months after the onset of the illness.

A biopsy of the skin, in January 1926, had disclosed mild intracellular edema of the rete Malpighii. The papillary bodies were edematous and infiltrated with lymphocytes. The cutis was edematous, and perivascular foci of lymphocytes were scattered throughout. In the deeper cutis the edema and infiltrations were most marked about the hair follicles and sweat glands. The elastica was only slightly fragmented. The collagen fibers were separated by edema. There were similar inflammatory foci in the upper panniculus adiposus.

In the muscle fibers there were swelling, loss of transverse striations, tortuosity, hyaline, granular and vacuolar degeneration and proliferation of the sarcolemmic nuclei. Fairly dense perivascular infiltrations of lymphocytes without vascular changes were present, also edema and infiltration of lymphocytes into the interstitial connective tissues. The connective tissue gave a minimal reaction.

Case 3.—A married woman registered at the clinic in 1923, complaining of inability to perspire in the summers since 1918 and of an urticarial eruption appearing on the skin after hot baths. These troubles disappeared before the onset of the present illness. She reregistered in May 1930. Her past and family histories were irrelevant. She had had weakness in the arms, back, thigh, respiratory muscles and muscles of the jaw in the previous four years. Dull pains and tenderness on pressure had recurred in these parts periodically. She had noted atrophy of the muscles about her shoulders, arms and hips. She suffered constantly from exacerbations and remissions of these symptoms, and in addition, had difficulty in swallowing, dyspnea with a feeling of great weakness in the respiratory muscles, and occasional attacks of nausea and vomiting.

On examination in May 1930, the skin appeared thickened, tight and fixed to the subcutaneous tissues (edema). The muscles of the arms, back and calves felt doughy, edematous and wasted. All the muscles of the extremities were weak, and the reflexes were correspondingly decreased.

On reexamination in October 1930, the skin was softer; the muscles were hypotonic and atrophied. The accessory muscles of respiration were used excessively, and the pharyngeal muscles were obviously weak. Auscultation revealed many râles, crackles and harsh breath sounds throughout the thorax. The pulse rate varied from 80 to 120; the blood pressure was 108 systolic and 75 diastolic, and the temperature ranged between 98.4 and 99.2 F. Laboratory studies revealed nothing of significance. A culture from biopsy tissue by Dr. Rosenow produced staphylococci. Another culture was made from the nasopharynx; on intravenous injection into rabbits this produced weakness and pain in the muscles; after death, yellow spots, smears from which contained diplostreptococci and staphylococci were found in the muscles of the animals. Four positive transfers to animals were accomplished.

A biopsy from the skin of the back (fig. *1A*) in May 1930 revealed no significant changes in the epidermis, but there were edema of the papillaris, subpapillaris and cutis, mild perivascular infiltrations of lymphocytes and swelling of the endothelial cells of the capillaries. The arrectores pilorum were infiltrated with lymphocytes. In the subcutaneous tissues there was a mild perivascular and slight diffuse infiltration of lymphocytes, with a few leukocytes and plasma cells.

A biopsy from the left vastus internus muscle (fig. *1 B* and *C*) revealed marked degeneration and atrophy of the muscle fibers. Many fibers presented hyaline and vacuolar degeneration, fragmentation, tortuosity and nuclear proliferation. Striations were frequently indistinct or absent. There was a moderately diffuse infiltration of the muscular tissues and about the vessels by lymphocytes, a few connective tissue cells and mast cells. A biopsy of muscle from the left deltoid in

November 1930 disclosed marked lymphocytic infiltrations and degeneration of muscle fibers. In May 1931 mucus began to collect in the patient's throat; she became severely dyspneic, then, suddenly, cyanotic; she was placed in an oxygen chamber but died within twelve hours.

At necropsy, on May 15, 1931, chronic myositis with muscular atrophy, organized bronchopneumonia and chronic tuberculosis of the right lung were found; other changes were not significant. Microscopically the skeletal muscles, including the diaphragm and the psoas, deltoid and intercostal muscles, presented marked parenchymal degeneration and lymphocytic infiltration. There was some proliferation of connective tissue but no diffuse changes in the vessels.

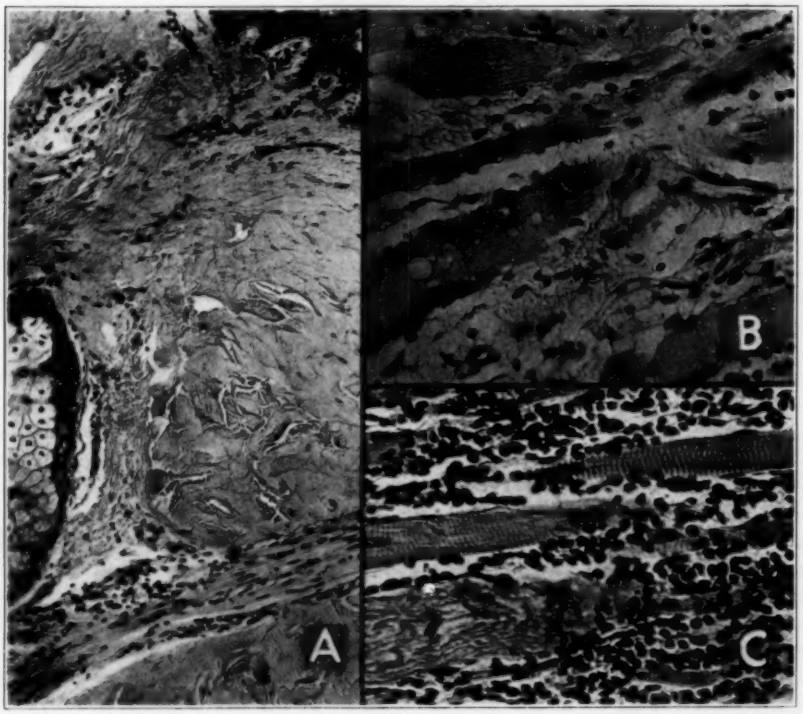

Fig. 1 (case 3).—Dermatomyositis. This figure is representative also of cases 1 and 2: *A*, infiltration of lymphocytes about subpapillary vessels and rather marked infiltration of lymphocytes into arrectores pilorum muscles; × 100. *B* and *C*, collagen homogenized; marked infiltration of muscle tissue, with swelling, vacuolar degeneration and loss of cross-striations in different muscle fibers (hematoxylin and eosin; *A*, × 100; *B*, × 250; *C*, × 275.

Case 4.—A farmer, aged 36, registered at the clinic in November 1933. His past and family histories were not relevant. When he had resumed work in May 1933, after a winter of idleness, he had found that his muscles were sore and tender on pressure. After two days he had been forced to give up physical effort. In June his knees, elbows and ankles became painful and stiff. A peculiar redness appeared at the suprasternal notch, and the skin of the abdomen became hard and thick. His forehead seemed stiff at times. His throat was sore during

the greater part of the summer. When he was about all day, his arms and legs felt hard at night.

An examination on Nov. 9, 1933, revealed a generalized increase in pigmentation. The face was slightly puffy. The skin over the clavicles and upper middle portion of the thorax was indurated, tense, shiny and livid red; it was indurated over the entire trunk but softer over the face and the peripheral portions of the extremities. Although the skin of the abdomen was thick, hard and tense, there was a peculiar circular area of softening about the umbilicus. The muscles revealed no signs of atrophy, but tenderness on pressure was elicited over the hamstrings,

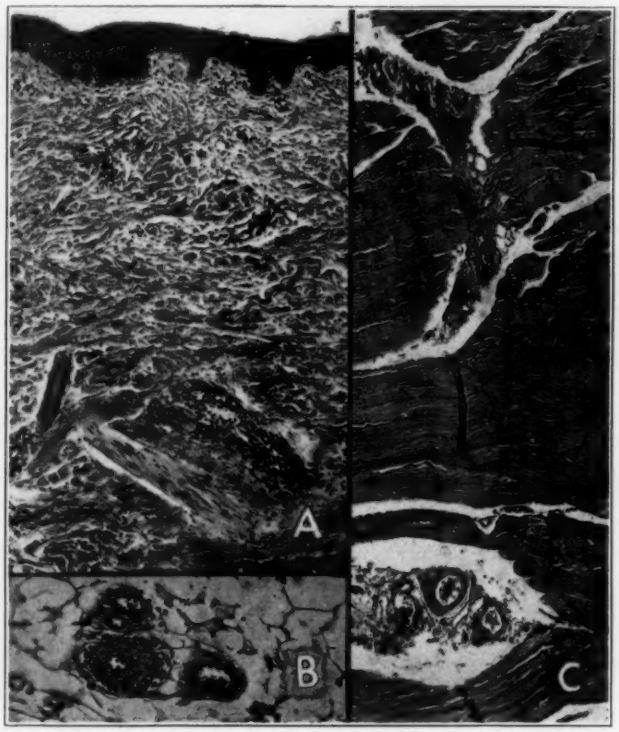

Fig. 2 (case 4).—Diffuse scleroderma. *A*, bundles of collagen separated by edema; perivascular infiltrations of lymphocytes and plasma cells throughout the cutis; × 65. *B*, thickening and lymphocytic infiltration of vessel walls in the subcutis; × 90. *C*, distinct perivascular infiltrations of lymphocytes and plasma cells in the interstitial tissues of the muscle; × 45.

calves, arms and forearms. The arms could be elevated only to the horizontal position, and the thighs could not be flexed more than about 110 degrees. The vessels of the extremities were not vasospastic. The blood pressure was 112 systolic and 68 diastolic; the temperature was 99 F., and the pulse rate, from 80 to 105. Roentgenograms of the joints revealed them to be normal. The basal metabolic rate was minus 1 per cent. All other laboratory studies and search for foci gave negative results.

Skin (fig. 2*A*) and muscle were taken from the right pectoral region for biopsy. Microscopically, the epidermis was unchanged: The bundles of collagen

throughout the cutis were separated by edema. The elastica was somewhat fragmented. Mild perivascular infiltration of lymphocytes and plasma cells was noticeable throughout, but was most marked in the deeper cutis and in the cutis-subcutis zone (fig. 2 *B*). The vessels were moderately swollen and infiltrated by lymphocytes. The epidermal appendages and cutaneous nerves were surrounded by a zone of edema and cellular infiltration. The muscle fibers were somewhat edematous (fig. 2 *C*), the cross-striations being a little indistinct. Definite perivascular infiltration of lymphocytes and plasma cells was found in the interstitial tissues. The walls of the vessels were slightly thickened and infiltrated by lymphocytes.

Treatment for one month with typhoid vaccine intravenously every second or third day produced ten or twelve chills; later, with the subcutaneous injection of a proprietary muscular extract there were no symptomatic or objective effects.

Case 5.—A farmer's wife, aged 50, had borne seven children; one child had died at birth; the others were living and well. She had had tonsillitis as a child, pleurisy at 17 and influenza in 1919. The illness because of which she finally had come to the clinic had begun with paronychia in April 1921, followed by aching and soreness of the fingers and feet and difficulty in walking. In June the muscles became weak and stiff, and she noted a generalized swelling. By November she had become bedridden, and movement in general was difficult and limited. She seemed to be muscle-bound. Swallowing became difficult. She had been feverish for three months. By April 1922 the swelling had disappeared, and some symptomatic improvement had occurred, although she remained helpless, and her skin was shiny and immobile. She was stiff and weak, and contractures of the arms and legs were present.

On examination, in November 1922, the skin was hidebound, thickened, tense and shiny. Subcutaneous hard plaques could be palpated on the medial surfaces of the thighs and in the region of the flexors of the elbows. Movements of the joints were severely limited by atrophied, firm fibrotic muscles; the deep reflexes were lost.

Repeated urinalyses gave negative results. The hemoglobin was 68 per cent; erythrocytes numbered 4,460,000, and leukocytes, 12,400 per cubic millimeter of blood; the Wassermann reaction of the blood was negative. No arsenic was found in the urine. The basal metabolic rate was plus 3 per cent on repeated examinations. Roentgenograms disclosed calcified nodules in the muscles and subcutaneous tissues of the thighs, arms and head. Both hands and elbows disclosed slight periarticular arthritis.

After twenty-three days in the hospital, the patient became nauseated, vomited and complained of severe epigastric pain. The abdomen was distended and tympanitic. A few days later the temperature rose to 101.2 F., and, on December 18 the pharynx filled with mucus; the patient became cyanotic and died.

At necropsy, on December 18, the following gross pathologic changes were found: generalized fibrosis of the subcutaneus tissues with local deposits of lime salts, generalized atrophy of the muscles with fibrosis, fibrosis of the myocardium, terminal edema of the glottis and bilateral hydrothorax (1,500 cc.) with anasarca.

Microscopic examination disclosed extreme diffuse increase of connective tissue in the heart, with hypertrophy of the muscle fibers. There was an increase of connective tissue about the larger vessels in the lungs, in the pulp of the spleen and its trabeculae and also in the periportal tissues of the liver. There were no other changes of note in the internal organs.

Fig. 3 (case 5).—This figure is representative also of case 6. Scleroderma with calcification. *A*, bundles of collagen hypertrophied and homogenized throughout the cutis; in the deeper cutis the vessel walls are thickened, hyaline and mildly infiltrated; × 65. *B*, sclerotic plaque in subcutaneous tissue showing hyaline and calcareous degeneration; × 65. *C*, an organized vessel, a comparatively normal vein and a thickened artery in a somewhat sclerotic interstitium; the muscle fibers on each side are hyaline; × 65. *D*, a dense focus of lymphocytes alongside a nerve in the interstitium of muscle; × 110.

Sections of the skin and muscle (fig. 3) taken before and after death presented the following changes:

Skin: The epidermis was moderately atrophic (fig. 3 *A*). The bundles of collagen were mildly hypertrophic and homogeneous throughout the cutis. The capillaries of the subpapillary network and the vessels of the cutis exhibited endothelial swelling and proliferation, thickening and hyalinization of their walls, in some instances complete occlusion as well as perivascular fibrosis, and small foci of lymphocytes. The hair follicles and sweat glands were slightly atrophic. The elastica was fragmented and apparently destroyed in certain fibrous areas in the cutis, especially about the hair follicles and cutaneous appendages. Elsewhere it was only apparently increased; no new fine fibers were found. The cutis appeared to be passing into an atrophic stage. The panniculus adiposus (fig. 3 *B*) contained diffuse and perivascular infiltrations of lymphocytes, small mononuclear leukocytes, plasma cells, fibroblasts and occasional mast cells. There was hyperplasia of connective tissue with sclerosis, fat alveoli being replaced by fibrous septums and degenerated hyaline sclerotic plaques with central deposits of calcium in masses and granules. The vessels were thickened and revealed endothelial swelling and proliferation with organization, leading to various degrees of obstruction in the lumens.

Muscle: In the fibers of muscle (fig. 3 *C* and *D*) there were great variations in size, hyaline degeneration, indistinctness of cross-striations, fibrous replacement of tissue and nuclear proliferation. The perimysium and endomysium were thickened and sclerotic (fig. 3 *C*), the interstitial tissue throughout showing the same change. The vascular changes were varied and marked (fig. 3 *C*), just as in the subcutaneous tissues. In the thicker degenerated hyaline plaques there were deposits of calcium. There was a perivascular and perineural (fig. 3 *D*) infiltration of plasma cells and lymphocytes in dense foci.

Case 6.—A farmer's daughter, aged 21, came to the clinic in September 1925 complaining of stiffness and extreme muscular weakness. In March 1923 she had had a severe attack of influenza with pneumonia. The illness had begun in August with specks before the eyes, soreness of the finger-tips and blotchy redness of the eyelids, ears and knuckles. Soon the hands, face and feet had become swollen, and all the muscles, weak, stiff, numb, hard and atrophic. She had had localized burning and creeping sensations, sharp pains on movement, dyspnea on exertion and transient difficulty in swallowing. She had lost 20 pounds (9 Kg.).

On examination, in September 1925, the patient's weight was 96 pounds (43.5 Kg.), and her height, 5 feet, 5 inches (165.1 cm.). There were ruddiness and puffiness of the hands and face and erythema of the eyelids, neck and extensor surfaces of the extremities. The muscles of the pectoral and pelvic girdles were atrophied, and there were resultant cordlike contractures about the shoulders and hips. Weakness and limitation of movement were evident at all the joints. The muscles were indurated, and there was a hard mass under the skin of the right thigh posteriorly. Roentgenograms revealed multiple areas of calcification throughout the subcutaneous and muscular tissues of the thighs and calves. All the laboratory examinations gave normal results.

The patient was admitted to the hospital on October 16. Nine days later she became nauseated and vomited. The temperature rose on succeeding days. The upper portion of the abdomen was tympanitic and the stomach distended; there was tenderness around the umbilicus. Signs of tetany appeared; the patient became cyanotic and died.

Necropsy disclosed atrophy and contracture of the muscles, deposits of calcium in subcutaneous tissues and intermuscular septums, atony of the stomach with

duodenal ileus, congestion and hemorrhages into the lungs, emaciation graded 2 and old healed pleuritis. Macroscopically the fibers of the cardiac muscle were shrunken and pale; the lungs were congested, and the spleen presented congestion and hyaline fibrinoid changes in the corpuscles. The walls of the vessels were thickened. The examination of other organs gave normal results.

Microscopic studies of the skin, subcutaneous tissues and muscles excised before and after death revealed atrophy of the epidermis. The collagen of the cutis was thinned and sclerotic in some areas, thickened, homogeneous and hyaline in others. The elastica was compressed, frayed and splintered. The appendages of the skin were atrophic. The vessels throughout showed endothelial swelling and proliferation, thickening and mantles of proliferating fibroblasts. In some of the vessels hyaline change was found, but perivascular lymphocytes and plasma cells were found in only a few.

In the subcutaneous tissue the fat was almost entirely replaced by bands of sclerotic, hyaline connective tissue with central masses of calcium deposit. The vessels were greatly thickened and hyaline, with sparse perivascular infiltration of connective tissue cells, lymphocytes and plasma cells. Some of the vessels were partially or completely occluded by organized fibrous tissue.

The changes in the muscles were marked. The perimysium and endomysium were thickened, sclerotic and hyaline, and contained calcareous masses. The vessels presented all the changes mentioned. There was marked hyaline degeneration of the muscle fibers; this was most severe immediately under the sclerotic sheaths. In some areas of more recent degeneration there were foci of mildly vascular granulation tissue.

COMMENT

The first three cases portray the clinical and pathologic features of dermatomyositis so well that a résumé of them is unnecessary. Indeed, the disease has been so well described by Steiner [1] that to describe it anew would constitute needless repetition.

In the literature [2] the terms "dermatomyositis" and "polymyositis" have been used, to some extent, more or less interchangeably. If it is understood that dermatomyositis is nothing more than a nonpurulent, nonhemorrhagic type of polymyositis in which there is, in addition, an inflammation of the skin, it immediately clarifies one's conception of the disease. Steiner defined the syndrome as an acute, subacute or chronic disease of unknown origin, characterized by vague and indefinite prodromes, followed by edema, dermatitis and inflammation of multiple groups of muscles.

The etiology has remained obscure,[3] and although toxemia, infestation with Gregarinae or Sarcosporidia, complications of infectious

1. Steiner, W. R.: Dermatomyositis: with Report of the Case Which Presented a Rare Muscle Anomaly but Once Described in Man, J. Exper. Med. **6**:407, 1904.

2. Weber, F. P., and Gray, A. M. H.: Chronic Relapsing, Polydermatomyositis with Predominant Involvement of the Subcutaneous Fat (Panniculitis), Brit. J. Dermat. **36**:544 (Dec.) 1924.

3. Schwarz, Richard: Beitrag zur Aetiologie der Polymyositis, Frankfurt. Ztschr. f. Path. **25**:141, 1921.

fevers, rheumatism and even tuberculosis [4] and syphilis have been considered by certain authors, the majority of workers have regarded bacterial infection as the cause. In a few instances streptococci have been cultured from involved tissues. Dr. Rosenow has been the first to reproduce the disease in animals. He isolated streptococci from cultures in case 2 and diplostreptococci and staphylococci from those in case 3.

The complications are interesting, not only because they are the immediate causes of death, but also because in most instances they resulted directly from the involvement of the muscles of deglutition, of respiration and less frequently of the myocardium. Of seventy-five undoubted cases gathered from the literature, forty were fatal. In fourteen of these, the cause of death was bronchopneumonia; in five, respiratory failure; in five, suffocation; in four, cardiac failure; in two, septicemia, and in two, tuberculosis. The prognosis is poor, 55 per cent of the patients having died within two years of the onset. The disease has been fatal in eight days and in two and a half years. The course of the disease is characterized by exacerbations and remissions. Any exacerbation may be severe and fatal. Although the greater number of patients are between 20 and 40 years of age, the mortality rate is relatively higher among the young and the old.

Treatment has been singularly unsuccessful, largely symptomatic and in most instances only palliative. Because bacteria may be the cause, foci of infection should be removed when present and when removal is feasible. Heat in various forms has been used locally. A series of foreign protein reactions produced by typhoid vaccine, given intravenously, has seemed to me to aid at least in securing remissions. Glycine was used in one case without effect.

Dermatomyositis must be distinguished from trichinosis [5] (Hepp [6] called his first case pseudotrichinosis), polymyositis hemorrhagica, various forms of purulent polymyositis, neuromyositis, poikilodermatomyositis, scleredema adultorum (Buschke), lupus erythematosus disseminatus and, rarely, tuberculosis and syphilis. These diseases are relatively easily distinguished from each other. The great problem is met in certain early cases of diffuse scleroderma which begin acutely or subacutely (table).[7]

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4. Grunke, W.: Tuberkulose als Ursache einer Dermatomyositis, Ztschr. f. klin. Med. **102**:311, 1925.

5. Dragoewa, N.: Polymyositis acuta und Trichinose, Berl. klin. Wchnschr. **56**:316 (April 7) 1919.

6. Hepp, P.: Ueber Pseudotrichinose, eine besondere Form von acuter parenchymatöser Polymyositis, Berl. klin. Wchnschr. **24**:297 (April 27); 322 (May 2); 389 (May 30) 1887.

7. Rake, Geoffrey: On the Pathology and Pathogenesis of Scleroderma, Bull. Johns Hopkins Hosp. **48**:212 (April) 1931.

The chronic atrophic type of diffuse scleroderma, with typical changes in the skin and muscles, is a familiar picture. Diffuse scleroderma is not limited to the skin and subcutis, but is primarily a disease of the connective and vascular tissues throughout the body. The muscles are frequently involved, and this involvement may be quite

Comparison Between Dermatomyositis and Scleroderma

	Dermatomyositis	Scleroderma
Etiology	Infectious	Infectious Vasomotor-trophic {endocrine / neurogenic[7]
Tissues involved	Skin and skeletal muscles sites of predilection	Connective tissues and vessels throughout body
Microscopic pathologic changes	Inflammatory infiltrations (a cardinal feature); parenchymatous degenerations of muscle, varied and diffuse (a cardinal feature); vascular changes, minimal; changes in connective tissues, minimal	Onset characterized by mild interstitial inflammation with edema (cf. dermatomyositis); later, inflammatory infiltrations, usually minimal; parenchymatous degenerations of muscle uniform and proportionate to interstitial sclerosis; vascular changes (a cardinal feature); changes in connective tissues; sclerosis (a cardinal feature)
Onset	Frequently acute or subacute, with prodromes and fever	Rarely acute or subacute, with prodromes and fever
Erythema and edema of the skin	Frequent early sign (face first in 25 per cent)	Occasional early sign (face in 12.5 per cent)
Situation of early involvement	Skin and muscle; face, back and proximal portions of the extremities (almost constantly)	Skin, muscle, vessels, nerves and joints: face and peripheral portions of the extremities (60 per cent)
Muscular pains and tenderness	Constant and characteristic	Inconstant but characteristic
Distribution of muscular involvement	Tending to be centrifugal (all or any groups of muscles may be involved)	Tending to be centripetal (all or any groups of muscles may be involved)
Neuralgia and rheumatoid pains	Transient prodromes	Common symptoms
Vasomotor disturbances	Never	Frequent
Pigmentation	Unusual and slight	Common and at times marked
Sclerodactylia	Never	Frequent
Trophic ulcers	Never	Not uncommon (over bony points)
Calcification	Not characteristic	Characteristic but infrequent
Course	Exacerbations and remissions	More uniform
Prognosis	Fifty-five per cent of patients die within two years	Usually chronic

independent of any change in the overlying skin or subcutis.[8] Three stages characterize the course of the disease: edema, induration and atrophy. Hence, in cases in which the disease begins acutely, the early combinations of slight fever, general malaise, edema and erythema of the skin and muscular pains and tenderness may confuse the clinical picture with that of dermatomyositis. Histopathologic studies of sec-

8. Thibierge, Georges: Contribution à l'étude des lésions musculaires dans la sclerodermie, Rev. de méd., Paris **10**:291, 1890.

tions of the skin and subcutaneous tissue [9] and of muscle [10] taken at this time may only add to the confusion; in scleroderma the earliest changes in these tissues consist of edema, mild perivascular infiltrations with lymphocytes, plasma and mast cells, swelling of the endothelial cells lining the vessels, and mild infiltration of the walls of the vessels with lymphocytes; also the parenchyma of the muscle may be edematous and the cross-striations of the individual fibers indistinct. Unfortunately for the diagnostician these are the changes found in early cases of dermatomyositis. Later, when induration and subsequently sclerosis render the clinical picture of scleroderma characteristic, the pathologic picture also becomes distinctive. Inflammatory infiltrations become less pronounced, are perivascular and may be present only in small foci. The vessels become thick walled; their lumens may be narrowed in all degrees even to occlusion with recanalization. There is great proliferation of connective tissue fibers with subsequent sclerosis and atrophy in the cutis, subcutis, perimysia and endomysia. In these situations sclerotic plaques may be found which may undergo hyaline and subsequently even calcareous degeneration.[11] The parenchyma of the muscle undergoes secondary degenerative changes of a uniform type, usually hyaline, and these are in direct proportion to the degree of sclerosis of vascular and connective tissue. On the other hand, in dermatomyositis perivascular and diffuse infiltrations of lymphocytes and plasma cells become and remain to the end an outstanding feature in cutaneous, subcutaneous and especially muscular tissues. The degenerations of muscle fibers are marked, varied and dependent on the inflammatory process which is existent throughout them. The changes in the vascular and connective tissues are relatively minimal.

To illustrate this difficulty in differential diagnosis, of the cases reported by Oppenheim [12] in 1899 as examples of dermatomyositis, one turned out four years later to be typical scleroderma. Rosenthal and Hoffmann [13] had a similar experience with a case in which their original diagnosis had been dermatoneuromyositis.

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9. Gans, Oskar: Histologie der Hautkrankheiten (Diffuse Sclerodermie), Berlin, Julius Springer, 1925, vol. 1, p. 101.

10. Cassirer, Richard: Die vasomotorischtrophischen Neurosen, Berlin, S. Karger, 1912, p. 536.

11. Durham, R. H.: Scleroderma and Calcinosis, Arch. Int. Med. **42**:467 (Oct.) 1928. Oehme, C.: Ueber diffuse Sklerose von Haut und Muskelm mit Kalkablagerung, Deutsches Arch. f. klin. Med. **106**:256, 1912.

12. Oppenheim, H.: Zur Dermatomyositis, Berl. klin. Wchnschr. **36**:805 (Dec. 11) 1899.

13. Rosenthal, Curt, and Hoffmann, Heinrich: Ein Fall von Dermatoneuromyositis mit Ausgang in Sklerodermie, Klin. Wchnschr. **3**:115 (Jan. 15) 1924. Klingman, W. O.: Dermatoneuromyositis Resulting in Scleroderma, Arch. Neurol. & Psychiat. **24**:1187 (Dec.) 1930.

Cases 1, 2 and 3 are, as has been stated, characteristic of dermatomyositis. Case 4 is one of subacute diffuse scleroderma of six months' duration. It is of great interest because the process had progressed far enough to show, besides edema and erythema of the skin, a diffuse induration, especially marked over the trunk, which gave the clinical impression of scleroderma. Further, the course was steadily progressive rather than surging forward, with exacerbations. As to the pathologic changes, although edema and perivascular infiltrations were moderately prominent, the walls of the vessels of the subcutis in particular were thickened and infiltrated; this would be unusual in a parallel case of dermatomyositis. In spite of the perivascular infiltration in the interstitial tissues of the muscles, the minimal changes in the muscle fibers and the absence of diffuse infiltration militate against the usual picture of dermatomyositis. Hence, a diagnosis of diffuse scleroderma could be made in this case early enough to emphasize the differential features in the face of the apparent similarities.

Cases 5 and 6 are typical examples of advanced scleroderma with the additional feature of calcification. Characteristic observations were the profound sclerosis in the skin, subcutaneous tissues and interstitial tissues of the muscles, along with marked vascular changes, and the minimal perivascular and perineural infiltrations and secondary uniform degeneration in the parenchyma of the muscles. The deposits of calcium in hyaline, sclerotic plaques of fibrous tissue were a true but not common feature of the disease. Mild sclerosis was present in the internal organs. A case clinically identical with these, and presenting similar subcutaneous deposits of calcium, as shown by roentgenographic examination and biopsy of the skin, presented repeatedly normal values for blood calcium (10 mg. per hundred cubic centimeters) and for inorganic phosphorous (4 mg. per hundred cubic centimeters). The case was not presented because no biopsy of muscle was obtained.

Despite any closer relationship [14] to dermatomyositis which diffuse scleroderma may ultimately be shown to have, it has been considered in this study in its conventional relationship. Certain clinical and pathologic parallelisms cannot at present justify a common etiologic basis without bacteriologic proof, but they can at least serve to suggest that this particular type of scleroderma is of infectious origin.

In conclusion, dermatomyositis presents a more or less characteristic clinical and pathologic picture. There is valuable evidence to indicate its infectious nature. The greatest problem in diagnosis is to distinguish it from certain early cases of acute or subacute scleroderma of the diffuse type. These particular cases of scleroderma begin with changes

14. Allan, William: Dermatomyositis or Scleroderma? Report of a Case, Arch. Dermat. & Syph. **19**:265 (Feb.) 1929.

in the skin and muscles similar clinically and pathologically to those in dermatomyositis. However, the interstitial inflammation seen histologically in scleroderma assumes a plastic character that produces sclerotic plaques, particularly in the subcutis, with resultant "scleroderma" of the skin, and in the sheaths and interstitia of muscles, with subsequent myosclerosis, whereas in dermatomyositis, inflammatory infiltrations and a variety of degenerations of the parenchyma of muscles are the outstanding features to the end.

AMERICAN LEISHMANIASIS

FURTHER OBSERVATIONS

HOWARD FOX, M.D.
NEW YORK

On a recent trip to Peru to study verruga peruana, I saw some cases of cutaneous leishmaniasis and discussed this subject with numerous Peruvian colleagues. Following my visit to São Paulo, Brazil, in 1929, I realized that there was a form of cutaneous leishmaniasis which occurs mainly in the forest regions of the Americas, extending from Mexico to the northern part of Argentina.[1] This disease has been observed in every country of South America except Chile, though by far the greatest number of cases are found in the tropical forests of Brazil, Peru and Bolivia.

The type of leishmaniasis in question differs clinically from oriental sore in that it affects the mucous membranes of the nose and throat in perhaps 20 per cent of the cases and as a result may cause permanent and often severe deformity. The investigations of Noguchi, in 1925, by agglutination tests apparently showed that the American type of leishmaniasis differed from both oriental sore and kala-azar. He considered that the three types were caused by separate species of leishmaniae. His work was later confirmed by Kliger, though Ray, in subsequent investigations, cast doubt on the work of both Noguchi and Kliger. Further research will be needed to establish definitely the relationship of oriental sore and American leishmaniasis.

My main object in this communication is to stress the fact that American leishmaniasis has many local names in the various countries in which it exists, a list of twenty-three such names being given by Castro in a recent thesis on cutaneous leishmaniasis in Latin America. In Peru and Bolivia the Indian name "espundia" is popularly used for the disease in question. I discussed this term with physicians in Lima, all of whom agreed without reserve that it was merely a local name for the same disease that exists in the three Americas.

Even in Peru there are at least five other local names besides espundia for the same disease. If, therefore, espundia is merely a local name for an involvement which is widespread, why should American writers of textbooks continue to designate this important disease by one

1. Fox, H.: American Leishmaniasis, Arch. Dermat. & Syph. **23**:480 (March) 1931.

of its local names? It would be just as inconsistent to speak of scabies as the Philadelphia itch or the Hongkong itch. Before my visit to Peru I felt reasonably sure that espundia was a local name, and now in view of the unqualified statement of leading Peruvian physicians I feel certain of it. Incidentally I saw several cases of mucocutaneous leishmaniasis in Lima which were clinically similar to cases I had observed in Brazil.

There is another question on which I would not presume to express a definite opinion, namely, whether "uta" is merely another synonym for American leishmaniasis, or whether, as some Peruvian physicians think, it is a different type of disease. The latter opinion is held by Dr. Edmundo Escomel, with whom I had a long conference, and by a few others. The majority, however, did not seem to share this view. While mucocutaneous leishmaniasis (espundia) is seen in the tropical forest regions, uta occurs on the western slope of the coastal mountains. According to Escomel, cases of uta resemble oriental sore in that they do not involve the mucous membranes and run a shorter and self-limited course. I saw several such cases, in which active and crusted lesions and scars of the face of two years' duration were evident. Dr. Escomel admitted that uta could not be classed definitely as a separate entity by clinical observation alone. Various immunologic tests would be necessary to establish this contention.

A NEW CLEANSING CREAM

JOHN G. DOWNING, M.D.
BOSTON

Many dermatologists have favorite combinations which from experience they have found to be of value.

Darier [1] prepared the following compound which he considered practical not only for the skin but for the scalp:

	Per Cent
Powdered soap	40
Lard	40
Oil of almonds	20
Essence of geranium	4 drops

He also used this soap mixture for the treatment of acne, but I received frequent complaints that it separated, and that it was for the most part unpleasant to use. There is no question that the use of soap as a local agent is necessary in the treatment of acne; the disadvantage of most soaps is that they are so irritating when applied directly to the skin that they are frequently discarded by patients who should use them.

To overcome these disadvantages, in 1932 the following formula, based on the aforementioned compound, was prepared; the prescription has the advantages of the soap mixture but none of its irritating qualities:

	Per Cent
Stearic acid	20
Liquid petrolatum	5
Triethanolamine [2]	5
Coconut oil soap	40
Distilled water	25
Glycerin	5

It is made in the following manner:

Heat the stearic acid, liquid petrolatum and triethanolamine to 85 C. in a porcelain or glass container. Heat the distilled water and the glycerin to 85 C. in a separate porcelain or glass container. Maintain the heat at 85 C. and dissolve the coconut oil soap by agitation. Add the aqueous solution to the stearic acid mixture with slow but constant stirring. Remove the mixture from the source of heat and continue stirring until it is cool. Do not beat air into the cream.

1. Darier, J.: Précis de dermatologie, Paris, Masson & Cie, 1923, p. 941.

2. Maynard, M. T. R.: Triethanolamine: An Adjunct to Dermatologic Therapy, Arch. Dermat. & Syph. **25**:1041 (June) 1932.

The result is a smooth, pleasant mass which does not separate even in summer and which gives a good lather when mixed with water. I have used it on sensitive skins and as an adjunct in the treatment of acne vulgaris and acne rosacea. It is applied with a pledget of cotton and removed with lukewarm water. The only disadvantage that I have found is that up to the present time it is difficult in many places to obtain triethanolamine. I have tried having the formula made without the triethanolamine,[3] but the resulting compound is not so good as the complete formula. Local druggists, however, can easily obtain triethanolamine from one of the jobbing houses.

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3. Triethanolamine is an excellent emulsifying agent. It has no alkaline irritating effect and increases the penetration of the oil, thus insuring the mild desquamation which is useful in cases of acne. It helps to form a cream of a pure white color which is easily removed with water.

EARLY NINETEENTH CENTURY DERMATOLOGY AND THE BROTHERS MAHON

THEODORE ROSENTHAL, M.D.
NEW YORK

The beginning of the nineteenth century saw many revolutionary changes in medicine. The dermatologic subject which probably gave rise to the greatest controvery at the time was that embraced by the general term of parasitic diseases. Generations of conjecture regarding the cause of scabies ceased when Renucci, in 1834, first demonstrated Acarus scabiei at the Hôpital St. Louis. This was preceded by the amusing deception practiced by Galès in 1812; his organism proved to be the mite found in spoiled cheese. The modern knowledge of human ringworm infections dates from Schönlein's (1839) and Gruby's [1] (1841) independent researches into the nature of favus.

Favus is not the only contagious parasitic disease of the scalp. There is another group of diseases designated as tinea tonsurans (*teignes tondantes*), or ringworm of the scalp, which have had a far more involved history than favus.

Space does not permit a detailed account of the curious manner in which the term favus was deviated from its original disease concept or of the acrimonious disputes between the French school, represented by Alibert, Biett and Cazenave,[2] and the Englishmen, Willan and Bateman,[3] regarding the correct terminology.

It is safe to say, however, that the keen observers of that day had even less success in the treatment of these vexatious infections than they had with proper classification and nomenclature. The fate of the child with ringworm of the scalp a century ago was a sorry one. The only methods of treatment were cruel and inhuman. A favorite instrument was the calotte, which was a leather disk smeared with pitch and applied to the scalp. When it was removed with a brisk twitch, the adherent hairs were uprooted *en masse*. Other methods involved the use of irritating ointments and solutions which occasionally gave rise

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Read before the Section of Historical and Cultural Medicine of the New York Academy of Medicine, May 9, 1934.

1. Gruby: Compt. rend. Acad. d. sc. **18**:583, 1844.

2. Cazenave, P. L. A., and Schedel, H. E.: Manual of Diseases of the Skin, translated by Thomas H. Burgess, London, H. Renshaw, 1842.

3. Bateman, Thomas: A Practical Synopsis of Cutaneous Diseases, According to Dr. Willan, ed. 8, London, Longman [and others], 1836.

to severe constitutional reactions and, more rarely, to fatal sequelae. Treatment consumed months and sometimes years.

It is, therefore, interesting to learn that at this time the Administration of Hospitals of France had entrusted the treatment of these conditions to the two Mahon brothers, laymen, who, to judge from the report quoted in a later paragraph, were able to secure superior therapeutic results. Their method was a secret one, which they refused to divulge, but it involved no harsh measures and accomplished cures in what seemed miraculously short periods.

The following is an extract of a report made to the General Welfare Council in 1816 by one of its members, on the condition of the hospitals and asylums in Paris from Jan. 1, 1804 to Jan. 1, 1814:

> The treatment of ringworm at the central admission office of the hospitals in Paris was instituted by decree on Dec. 31, 1806, and a similar treatment commenced at the Infants' Hospital.
>
> Before adopting the remedy of the Mahon brothers the council had conducted a two year trial of it at the Hôpital St. Louis under the eyes of its own physicians; their report was favorable. From 1809 to 1813 the treatment was tried on 795 children! 527 were cured. One hundred and ninety-six were not cured, or, if they seemed to be, the disease recurred, and 72 were still under treatment on Dec. 31, 1813. The Mahon brothers received in addition to a yearly stipend of 1000 francs each, six francs per capita for the children declared cured.

This, then, was the confused and uncertain status of parasitic diseases during the first decades of the nineteenth century in France, when, in 1829, there appeared a handsome octavo volume entitled, "Recherches sur le siége et la nature des teignes," [4] by Mahon the younger, one of two brothers who, though laymen, were officially entrusted with the treatment of parasitic diseases in the hospitals of Paris, Lyons, Rouen, Dieppe, Elbeuf and Louviers.

The book, consisting of a lengthy introduction of forty pages, three hundred and seventy-five pages of text and five excellent colored plates drawn by Zwuinger, son-in-law of the elder Mahon, represents a significant milestone in the knowledge of parasitic diseases. It is the first volume to be devoted entirely to a specialized subject of this sort and considers thoroughly all of the aspects of the question in approved monographic style. The date, 1829, is noteworthy as it falls before the discovery of the specific agents causing the infections.

The foreword is a quotation from Alibert [5] indicating the desirability of more careful and exact research into the origin of *teignes,* or parasitic diseases of the scalp.

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4. Mahon: Recherches sur le siége et la nature des teignes, Paris, J. B. Baillière et fils, 1829.

5. Alibert, J. L.: Précis théoriques et pratique sur les maladies de la peau, Paris, Caille Rairer, 1817.

The preface contains a lengthy declaration of gratitude to the Administration of Hospitals of Paris for deigning to entrust the treatment of tinea to the Mahons, thereby providing them with the means of making thousands of observations of all types of this disease. They wish to thank, among others, Alibert, Richerand and Biett for assistance in their work.

In the introduction, the words of Alibert are quoted to show the incentive for undertaking the task. His diligence in pursuing his studies and, as a result, his ability to separate into other groups diseases which formerly were classified with the tineas are alluded to.

The classification followed by the Mahons is: "t. faveuse, t. tondante, t. amiantacée, t. furfuracée, t. muqueuse, t. granulée, crasse laiteuse, crasse membraneuse." A chapter is devoted to each species. Chapters on differential diagnosis and treatment, with tables showing the results of successful treatment, conclude the book.

Mahon the elder, according to Sabouraud,[6] discovered ringworm of the scalp and named it. His description, which was far more complete and explicit than that of Willan and Bateman, seems to fit the type of ringworm now called microsporosis. He described areas of various sizes which were denuded of hair, with broken-off hairs still visible; in the areas the skin was bluish, but when it was scratched it was covered with whitish dust. Not only is microsporosis described thoroughly, but mention is made of its frequent transmission to the glabrous skin and to the finger-nails. Trichophytosis and favus of the nails are described and identified as two separate entities, and the thickening and distortion of the nail and the difference in color (white in trichophytosis and yellow in favus) are emphasized. Thus the most important manifestations of the disease were simultaneously and precisely recorded by an observer who was not even a physician.

As a matter of interest, favus of the nails was discovered by one of the Mahon brothers, who accidentally infected himself while epilating a patient with his finger-nails. He was thus able to observe the course and evolution of the disease at first hand.

The chapter on treatment is wordy and replete with apologies; it mentions all of the treatments that have been employed except that of the Mahon brothers. Several histories of fatal accidents ensuing after treatment are reported. As to their own treatment, the authors merely state that it is simple, that it requires hospitalization and that large numbers of patients can be treated by a few men. They even compute the cost of treatment as 1.25 francs for each patient a day.

They state that a great day would dawn if they were able to divulge their secret, but because of sacred family duties and inviolable stipulations they cannot reveal the nature of it.

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6. Sabouraud, R. J. A.: Les teignes, Paris, Masson & Cie, 1910.

This, of course, was a necessary expedient because of the financial considerations involved. If the brothers had made public their methods, their only means of livelihood would have disappeared. Suffice it to say, however, the Mahons had realized early in their experience, the importance and usefulness of epilation; they became expert in the use of their finger-nails in the operation. Epilation with forceps had fallen into disuse after Chauliac, Pare, Astruc and Sauvage. The mysterious ointments and secret powders used by the Mahons were undoubtedly employed to put the inquisitive off the track.

The original work and discoveries of the Mahon brothers had surprisingly little reverberation. It is true that Alibert [7] alluded to tinea tonsurans, and several of his pupils named it, but there was evident confusion of terms and ideas; nevertheless, as a physician at the St. Louis Hospital, Alibert was well acquainted with the work of the Mahons. The majority of contemporary writers ignored tinea of the scalp, neither mentioning nor describing it. In historical sketches of the work in the Hôpital St. Louis, Brocque [8] and Hardy, made no mention of the Mahons, nor did Meunier.[9] Rayer [10] stated,

> Of all the depilatory methods proposed, that of the Messers. Mahon is unquestionably the best. They begin by cutting the hair, etc., removing incrustations with flour poultices . . . then the parts are annointed with the depilatory ointment; later an epilatory powder is used, in conjunction with a fine comb.

A few pages later, Rayer again mentioned the work of the Mahons and quoted from a report of the analysis of their proprietary remedies published by Bracconot. Three powders were employed, and their chemical constitution proved to be essentially an impure subcarbonate of potash.

In his report on the organism subsequently named Trichophyton endothrix, Gruby,[1] mentioned Mahon in the title of his paper.

Bateman [11] mentioned Mahon's method of removing infected hairs in favus. He also mentioned Mahon in his bibliography.

Bazin [12] repeatedly cited Mahon in a complimentary fashion. After stating that the younger Mahon deserves the honor of being the first to name and describe the entity of tinea tonsurans, he said that he could not understand on what basis the reproaches which Alibert and later Cazenave directed toward him were founded.

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7. Alibert, J. L.: Monographe des dermatoses, ou précis théorique et pratique des maladies de la peau, Paris, Daynac, 1832.

8. Brocque: Presse méd. **11**:101, 1903.

9. Meunier: Histoire de la médecine, Paris, J. B. Baillière et fils, 1911.

10. Rayer, P. F. D.: Treatise on Diseases of the Skin, ed. 2, London, J. B. Baillière, 1835.

11. Bateman,[3] p. 216.

12. Bazin, E.: Leçons théoriques et cliniques sur les affections cutanées parasitaires, Paris, A. Delahaye, 1862, p. 152.

In 1840, Cazenave [2] rediscovered tinea tonsurans; he gave a masterly clinical description of it and made the first differentiation of ringworm of the scalp from alopecia areata. It seems entirely probable that the diagnosis of ringworm of the scalp in France from 1830 to 1840 was made only by the Mahon brothers, laymen.

An intensive search for biographic data relating to the brothers has been disappointing. Contemporary and modern medical periodicals, as was to be expected, completely ignored them. Even at the Hôpital St. Louis, where the brothers worked for years, there is no information available as to their lives. Prof. Léon Brodier, the distinguished historian, has assured me that he is unaware of any document bearing on the brothers Mahon, "considered," to use his own words, "as charlatans by French dermatologists." The publishers, J. B. Baillière and son, the firm that brought out the Mahons' book over a century ago, have no knowledge of the Mahons.

Quérard [13] mentioned the younger Mahon in connection with the book and gave the date of his death as October 1833, at Paris. This apparently conflicts with other data supplied by Brodier,[14] who stated that the younger Mahon wrote another book in 1868, also published by Baillière, entitled "Considerations sur le traitement des teignes" which is listed incorrectly in the *"Index Catalogue"* under the name of Mignot-Mahon. I have been unable to obtain a copy of the book. Brodier stated that the younger Mahon had a son and two sons-in-law, called Vaconin and Mignot-Mahon, who also busied themselves with the treatment of ringworm. There was also a son-in-law of the elder Mahon, named Guilbert.

For many years the brothers Mahon and their family conducted a private clinic devoted to the treatment of ringworm infections on the Rue du Pas-de-la-Mule, Paris. The clinic was maintained by their descendants until 1914. One of the best known of these, Paul de Molènes-Mahon, who died in 1916, published a thesis on polymorphous erythema in 1884.

The spectacle of a nonmedical family attaining to special skill and ability in the diagnosis and treatment of a particular disease is not unusual in the history of medicine and is not limited to dermatology. An outstanding example is that of the famous Thomas family of bonesetters in Liverpool, who flourished at about the time of the Mahons.[15]

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13. Quérard: La France littéraire, Paris, 1827-1864, vol. 5.

14. Brodier, L. J.-L.: Alibert, médecin de l'Hôpital Saint-Louis (1768-1837), Paris, A. Maloine et fils, 1923; personal communications to the author.

15. Bick, E. M.: History and Source Book of Orthopædic Surgery, New York, Hospital for Joint Diseases, 1933.

Sabouraud,[6] who evinced a most friendly spirit to the Mahons, called them "empiriques" (empiricists or possibly lay healers). This term is less opprobrious than charlatan, employed by Brodier. Garrison,[16] in his recent paper on quackery, undertook to describe three types of medical impostors; speaking of the charlatan, he described him as "the Doctor Know-all of Grimm's Fairy Tales, whose top-heavy assumption of omniscience ranges anywhere from parade of erudition to maundering about the ultimate nature of disease." Not so with the Mahons; it is safe to say that, had they possessed the necessary professional qualifications, their book would have been hailed as a solid contribution to dermatology—a landmark of progress. It is true that by keeping their methods and remedies secret they violated the fundamental canons of medical ethics, but as they were not of the profession, they should be judged by more charitable and elastic standards.

More than a century has gone by since the time of the Mahons, and it does not seem proper that one should forget these brothers who, working for a generation, accepted by their illustrious contemporaries and officially certified by the government in their special capacities, secured genuine therapeutic successes in a field in which all others had encountered failure.

115 West Eighty-Sixth Street.

16. Garrison: Bull. New York Acad. Med. **9**:11 (Nov.) 1933.

POLYNEURITIS FOLLOWING NEOARSPHENAMINE THERAPY

REPORT OF A CASE WITH AN ASSOCIATED EXFOLIATIVE DERMATITIS

FREDERICK KELLOGG, M.D.
AND
NORMAN N. EPSTEIN, M.D.
SAN FRANCISCO

Polyneuritis as a complication of arsphenamine therapy is rare. We were able to find only 37 cases recorded in the literature. Recently we have observed a patient with polyneuritis and exfoliative dermatitis following a series of injections of neoarsphenamine.

REPORT OF CASE

A 46 year old iron-worker of German-American parentage entered the University of California Hospital on Feb. 20, 1933, complaining of weakness of the legs and pain in the feet of two months' duration. When he was 19 years old he had gonorrhea and a soft chancre. At that time enlarged inguinal glands were removed, and he was given inunctions of mercury. When he was 20 and also when he was 29 years old he was ill with daily chills and fever while living in regions infested with malaria.

Nine months before entry into the hospital he felt fatigued and had an occasional attack of feverishness which he thought was a recurrence of the malaria. Friends noted a yellowish cast to his skin. He consulted a physician and was given six weekly injections of neoarsphenamine of 0.6 Gm. each. The first injection was given on Oct. 14, 1932. Following the fourth injection he began to suffer with pruritus over the wrists and ankles. The day after the sixth injection swelling of the ankles, hands and face appeared, and transient amblyopia developed. The following day the patient became acutely ill with nausea and vomiting. A severe exfoliative dermatitis then appeared, which kept him bedfast for a month. During this time he lost his hair and noted changes in the growth of his nails. Three weeks after the onset of this acute illness he began to have constant pain in the left foot, which was intensified by weight-bearing. A month later similar pain appeared in the right foot. Numbness of the fingers of the hands was occasionally present.

The patient took alcohol infrequently. He had never had a Wassermann test of the blood.

Examination showed a well developed man presenting evidence of recent loss of weight. The skin was roughened; generalized brownish pigmentation was present, with small scattered areas of depigmentation; the skin over the soles was thickened. The hair was sparse on the body. The sclerae were brownish rather

From the Divisions of Medicine and Dermatology, University of California Medical School.

than icteric; the pupils reacted well to light and in accommodation; the fundi showed numerous pigmented spots suggestive of chorioretinitis; the visual fields were normal. The heart showed no enlargement to percussion or on roentgen examination; the cardiac sounds were muffled and distant. The blood pressure was 140 systolic and 90 diastolic. The liver and spleen were not felt. A scar was present on the shaft of the penis. All of the nails were rough and thickened over their distal portions. The muscles of the left leg, especially the extensor group of the foot, were weak and wasted. The left calf and the feet were tender to pressure. The ankles were slightly swollen. The patellar reflexes were active, but the achilles tendon reflexes were not obtained. Positional and vibratory sensation were not impaired.

Laboratory examinations showed a normal amount of hemoglobin and normal numbers of erythrocytes and leukocytes. The differential count showed 12 per cent monocytes. No malarial parasites were found. The icteric index, the rate of sedimentation of the red blood cells, and the determinations of the nonprotein nitrogen of the blood were within normal limits. The Wassermann test of the blood was negative, but the Kahn test gave the following readings: 1 plus, 2 plus and 3 plus, with an average of 2 plus.

An Addis concentration test of the urine showed a low specific gravity (1.017), an abnormal amount of albumin (200 mg. in twelve hours), a normal number of casts and one hundred times the normal number of erythrocytes, leukocytes and epithelial cells. Urobilinogen was present in normal amounts, according to the Wallace-Diamond test (a quantitative modification of Ehrlich's test). Lead was absent. The phenolsulphonphthalein excretion was normal. The result of a urea clearance test (Van Slyke) was at the lower limit of normal.

The rose bengal test of hepatic function showed 70 per cent retention of the dye in eight minutes and 45 per cent retention of the dye in sixteen minutes. The modified dextrose tolerance test showed marked hypoglycemia (38 mg. per hundred cubic centimeters) after two hours. Both of these tests indicated a damaged liver.

The results of an examination of the spinal fluid were entirely negative. An electrocardiogram showed an inverted T wave in the first lead. A roentgenogram of the left foot showed marked bony atrophy. Analysis of the hair on the scalp showed 0.075 mg. of arsenic trioxide per 100 Gm. of hair, which is not an abnormal amount.

Sodium thiosulphate (1 Gm. daily) was administered intravenously for four days, and the patient was given a diet with a high content of carbohydrates and vitamins. The feet were treated with alternate cold and hot applications, massage and active and passive exercise.

The symptoms in the feet steadily, though slowly, abated. Four months after the acute illness, however, numbness of the left hand became marked, and weakness of the extensors of the left wrist developed. These symptoms in turn gradually disappeared, and the patient was able to return to work nine months after the onset of illness. At this time the urinary findings were approximately the same as before, and the hepatic functions, as shown by the rose bengal test, had only moderately improved.

Because of the chorioretinitis, the penile scar and a single moderately positive Kahn test, a diagnosis of syphilis was made. It should be noted that the blood Wassermann test was negative on the first examination, which is not an unusual finding following an exfoliative dermatitis. Six months after the patient's entry into the hospital, without further treatment for syphilis, the Wassermann and Kahn tests of the blood gave readings that averaged 1 plus. The impairment of

the function of the liver and kidneys may have been due to syphilis, though neoarsphenamine often affects the liver and may rarely affect the kidneys. The exfoliative dermatitis and the polyneuritis were undoubtedly due to neoarsphenamine.

REVIEW OF THE LITERATURE

In 1920, Beeson [1] reported a case of polyneuritis and exfoliative dermatitis following treatment with neoarsphenamine and reviewed the literature up to that time. He was able to collect 21 additional cases in this way. We have found reports of 15 more cases [2] prior to and since that publication, making a total of 37 cases.

The low incidence of polyneuritis as a complication of treatment with arsphenamine is shown by the fact that Moore and Keidel [2b] observed only 2 instances in a series of 47,000 injections of the arsphenamines, although there were 21 cases of exfoliative dermatitis in the same group. Laurent [2f] reported that only 2 cases of polyneuritis occurred in 2,800 patients receiving a total of 27,000 injections of neoarsphenamine. In our experience with over 40,000 injections of the arsphenamines in the past eight years, the case reported is the only instance of polyneuritis which we have encountered.

Polyneuritis has followed the use of neoarsphenamine much more commonly than that of arsphenamine. Of 36 reported cases in which the type of arsphenamine used was stated, neoarsphenamine was used in 25 and arsphenamine in 11. In 1912, Wechselmann [3] stated that though

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1. Beeson, B. B.: Polyneuritis Plus Dermatitis Exfoliativa Following Neoarsphenamine, Arch. Dermat. & Syph. **2**:337 (Sept.) 1920.

2. (*a*) Wolff, A., and Mulzer, P.: Zur Kasuistik der Behandlung der Syphilis mit Neosalvarsan, München. med. Wchnschr. **59**:1706, 1912. (*b*) Moore, J. E., and Keidel, A.: Dermatitis and Allied Reactions Following the Arsenical Treatment of Syphilis, Arch. Int. Med. **27**:716 (June) 1921. (*c*) Solomon, N. I.: Case of Polyneuritis After Treatment with 606 and 914, Med. obozr. **79**:587, 1923. (*d*) Schamberg, J. F., and Greenbaum, S. S.: Multiple Neuritis with Jaundice After the Use of Neoarsphenamine, J. A. M. A. **84**:163 (Jan. 17) 1925. (*e*) Chabanier, H., and Sézary: Névrites et pseudo-tabes arsénobenzéniques, Bull. et mém. Soc. méd. d. hôp. de Paris **49**:279 (Feb. 20) 1925. (*f*) Laurent, C.: Six années de thérapeutique par les novarsénobenzènes, Ann. d. mal. vén. **20**:801, 1925. (*g*) Hassin, G. B., and Read, C. F.: Facial Diplegia in Neoarsphenamine Polyneuritis, J. Nerv. & Ment. Dis. **63**:352, 1926. (*h*) Cornil, L., and Pierson, E.: Erythrodermie et polynévrite arsénobenzolique, Bull. Soc. franç. de dermat. et syph. **34**:43, 1927. (*i*) Bériel, L., and Gaté, J.: Diplégie faciale survenue au cours d'un traitement novarsénobenzolique un mois après l'apparition d'un chancre syphilitique de la region génitale, Lyon méd. **141**:465, 1928. (*j*) Olivet, A.: Polyneuritis nach Neosalvarsanbehandlung bei syphilitischen Ikterus, Deutsche Ztschr. f. Nervenh. **123**:288, 1932. (*k*) Urechia, C. I., and Dragomir, L.: Polynévrite arsénobenzolique, Bull. et mém. Soc. méd. d. hôp. de Paris **48**:825, 1932.

3. Wechselmann: Ueber Neosalvarsan, München. med. Wchnschr. **59**:2099, 1912.

polyneuritis may follow large doses of neoarsphenamine it is relatively rare after administration of arsphenamine. Sicard [4] showed that the patellar and achilles tendon reflexes commonly disappear after injection of large amounts of neoarsphenamine. Even after a course of only 3 or 4 Gm. of neoarsphenamine diminution in these reflexes occurred. Sicard felt that such changes in the achilles tendon reflex should warn against continuing this therapy. Schamberg and Greenbaum [2d] explained the greater incidence of polyneuritis after injection of neoarsphenamine than after that of arsphenamine on the basis of the greater instability of neoarsphenamine, with the formation of toxic oxidation products. Olivet [2j] believed that neoarsphenamine had a neurotropic toxic action which was usually counteracted by the liver and that, therefore, in patients with impaired hepatic function, polyneuritis might occur more readily. Cornil and Pierson [2h] also suggested that hepatic insufficiency plays an important rôle in the toxic effect of the arsphenamines.

We have found no report of a case in which polyneuritis followed the use of sulpharsphenamine, silver arspehenamine, bismarsen or tryparsamide. Chabanier and Sézary [2e] reported 4 cases following the use of eparseno, an organic pentavalent arsenical.

In a few of the reported cases, chronic alcoholism may have been a predisposing factor in the development of polyneuritis.

CLINICAL PICTURE

The reported cases were equally distributed in the two sexes, and age did not seem to be a predisposing factor.

Polyneuritis following treatment with arsphenamine usually develops several weeks after the drug has been administered, although occasionally it may appear within a few days. The condition is not dependent on the quantity of arsphenamine given, as only a few cases have been reported in which an excessive amount was administered. As a rule, a few injections are sufficient to produce polyneuritis in susceptible persons, and in 2 reported cases the condition developed after a single dose of the drug, 0.6 Gm. in one instance and 0.45 Gm. in the other. These 2 patients had been inadequately treated previously.

Paresthesias are usually the initial symptoms. These are followed by dull pain, then weakness and finally flaccid paralysis of the affected parts. Sicard [4] showed that a diminution or an absence of reflexes may precede the subjective symptoms. The symptoms gradually increase in intensity over a period of several weeks or months and then gradually

4. Sicard, J. A.: Traitement de la syphilis nerveuse, Presse méd. **28**:281, 1920.

disappear. Complete recovery may require from two to six months. Occasionally, as in the case reported here, transient exacerbations occur.

The extensor muscles are chiefly affected. The lower extremities are almost always involved, and in about 50 per cent of the cases the upper extremities are also involved. Rarely a single limb is affected. Occasionally a unilateral, more rarely a bilateral, facial paralysis occurs.

Arsphenamine polyneuritis is usually associated with other toxic manifestations of this drug which antedate the development of the polyneuritis. In 14 of the reported cases a severe exfoliative dermatitis was present. In 6 cases jaundice developed. Three patients had marked gastro-intestinal symptoms. In 3 patients signs of nephritis developed.

If other causes of polyneuritis can be excluded, the diagnosis is easily made. While syphilitic polyneuritis may rarely occur, it responds readily to specific therapy. Because of the paresthesias, pains in the extremities and diminution or absence of reflexes, tabes dorsalis may be erroneously diagnosed. Tabes, however, is readily eliminated, as Chabanier and Sézary [2e] have pointed out. In tabes the paresthesias and pains are not limited to the feet and hands to the extent that they are in polyneuritis. In polyneuritis, as contrasted with tabes, the pupillary reflexes are normal, the patellar reflexes are usually present, and the sphincters are not involved. In tabes, radicular bands of altered sensation can often be demonstrated, whereas sensory changes in polyneuritis occur mainly in the peripheral nerves. Finally, in polyneuritis, examination of the spinal fluid shows a normal gold curve.

COMMENT

The polyneuritis in our patient was not so severe or so extensive as that described in other reports in the literature. However, recovery was slow, nine months elapsing after the onset of the illness before the patient was able to return to his work. He had exfoliative dermatitis and showed impaired hepatic function. These complications have been common in other cases of polyneuritis following the use of the arsphenamines. If the views of Olivet and Cornil and Pierson are correct, the hepatic insufficiency was an important etiologic factor in our patient's polyneuritis.

The long interval usually occurring between the administration of an arsphenamine and the onset of polyneuritis is noteworthy and comparable to the delays noted in other reactions following arsphenamine therapy.

It is interesting that polyneuritis is much less frequent following the use of arsphenamines than following the use of inorganic arsenicals. Osborne [5] suggested that the differences in toxic effects of various

5. Osborne, E. D.: Microchemical Studies of Arsenic in Arsenical Dermatitis, Arch. Dermat. & Syph. **18**:37 (July) 1928.

arsenicals are dependent on the valence of the arsenic. Using a microchemical technic, he studied the localization of arsenic in the skin following the administration of arsphenamine (trivalent arsenic) and of pentavalent inorganic arsenicals. When arsphenamine was given, he found depositions of arsenic predominantly about the small arterioles and capillaries of the corium, whereas when inorganic pentavalent arsenicals were given he found the depositions chiefly in the ectodermal structures, such as the epidermis, the sudoriferous and sebaceous glands, the hair follicles and the hair. He concluded that trivalent arsenic is primarily a vascular toxin, while pentavalent arsenic is primarily an ectodermal toxin and may therefore more readily affect the nervous system.

Osborne pointed out, however, that his work does not exclude the influence of the benzene rings with attached amino and hydroxyl groups which are present in arsphenamine. Probably this organic structure of arsphenamine is more important than the valence in determining its toxic action. Tryparsamide (pentavalent arsenic) more commonly causes optic neuritis than arsphenamine (trivalent arsenic). This, however, may be explained by differences in organic structure of the compounds rather than by the valence of the arsenic. Thus Young and Loevenhart [6] showed in experiments on rabbits that optic atrophy usually followed trivalent and pentavalent organic arsenicals with an amino group in the *para* position, whereas it did not occur if the amino group were in the *ortho* or *meta* position.

SUMMARY

1. A case is reported of exfoliative dermatitis and polyneuritis following neoarsphenamine therapy.

2. Analysis of the reported cases shows polyneuritis to be a rare complication of neoarsphenamine therapy and an even less frequent complication of arsphenamine therapy. This complication generally occurs in association with other complications of arsphenamine therapy. The onset of the neuritis is usually delayed, and though the prognosis is good, recovery is slow.

6. Young, A. G., and Loevenhart, A. S.: The Relation of the Chemical Constitution of Certain Organic Arsenical Compounds to Their Action on the Optic Tract, J. Pharmacol. & Exper. Therap. **23**:107, 1924.

SUPERFICIAL PUSTULAR FOLLICULITIS OF THE NEW-BORN

S. IRGANG, M.D.
AND
E. R. ALEXANDER, M.D.
NEW YORK

A superficial, benign type of pyodermia occurring in the new-born has been observed, rather infrequently, during the past few years in the obstetric service of Harlem Hospital. In this institution the vast majority of patients are Negroes, which probably accounts for the fact that the infection has been observed entirely in the Negro infant. It is likewise possible that this cutaneous disorder is more common in this race.

Except for a paper by Reed,[1] a search of the literature failed to reveal a discussion of this disorder in the new-born. Reed discussed some aspects of the condition, but he was somewhat confusing since he conveyed the impression that this type of pyodermia is a clinical variation of impetigo contagiosa neonatorum. Since this superficial, benign pyodermia is at times confused with impetigo contagiosa of the new-born, it seems appropriate to stress the importance of the superficial pustular folliculitis of the new-born not only from the standpoint of diagnosis but also from the prognostic and therapeutic points of view.

The condition is a vesiculopustular dermatitis occurring within the first week of life. No fatality was observed in any case at the Harlem Hospital. None of the patients showed evidence of the disease at birth. The infant with this condition is well nourished and in apparent good health. Its weight is normal, and it does not perspire excessively. The dermatitis appears suddenly without apparent cause, the lesions appearing simultaneously over different parts of the cutaneous surface. It is unusual for new lesions to make their appearance after two or three days. Their number varies widely; they are superficial and nonpruritic and are most numerous on the face, chest and abdomen. The infant does not appear to be affected by the cutaneous disorder. The temperature remains practically normal throughout.

The chief type of lesion is the vesicle, which is transformed, in many instances, to a pustule after a short time. In some cases a small number of papules are observed at the outset; these either continue as such until

From the Departments of Dermatology and Obstetrics, Harlem Hospital.

1. Reed, C. B.: Pyodermatitis Neonatorum, Arch. Dermat. & Syph. **18**:26 (July) 1928.

resolution or are transformed into vesicles. The lesions are discrete in the vast majority of instances and vary in size from that of a pinhead to that of a split pea; there is no tendency toward grouping.

The thin vesicular wall is either slightly elevated or flattened, and on rupture a small amount of clear, colorless serous substance is evacuated. An erythematous areola of varying extent and intensity surrounds the lesions; in some cases lesions without an inflammatory base are seen. As the dermatitis begins to resolve, the vesicles and pustules become capped by a small crust, while a scale appears on the resolving papule. Resolution takes place within from seven to ten days, leaving pigmentation but no scarring.

In an occasional case, a complicating mild type of furunculosis appears. In none of the patients was it necessary to resort to surgical measures to effect a cure. While this complication is not serious, it tends to delay recovery.

Unfortunately, bacteriologic examination was made in only one case. The contents of a number of vesicles were cultured, and Staphylococcus albus was the only bacterium isolated. Since Staph. albus is a constant inhabitant of the skin and since it is normally but slightly pathogenic, it is possible, in unusual instances, for the infant to exhibit temporarily, in the first week of life, a lowered resistance to this type of organism.

Despite the fact that this pustular folliculitis of the new-born does not appear to be transmitted by contact, rigid isolation of infected persons should be strictly enforced, since these patients are usually cared for in the obstetric service. Baths with soap and water twice daily were the only treatment required to effect a cure. In no case was it necessary to resort to local antiseptic lotions or ointments.

The two principal diseases which must be considered in differential diagnosis are dermatitis medicamentosa (iododerma and bromoderma) and impetigo contagiosa neonatorum. An eruption due to iodide or bromide can readily be excluded by the history of medication in either mother or child. The following is a summary of the differential characteristics of impetigo contagiosa and superficial pustular folliculitis of the new-born:

Impetigo Contagiosa	Superficial Pustular Folliculitis
1. Presence of vesicles and bullae which rupture rapidly with or without formation of crusts; no papules.	1. Presence of vesicles and pustules which persist intact for a much longer time; beginning resolution indicated by crusts; papules sometimes seen.
2. Appearance of new lesions until local antiseptics are applied.	2. Cessation of new lesions after two to three days without resorting to antiseptics.
3. Spreads by contact.	3. Contact apparently has no effect.
4. Cured by application of local antiseptics.	4. Cured by baths with soap and water.

SUMMARY

1. Superficial pustular folliculitis of the new-born has received but scant attention from the dermatologist. At times it has been confused with impetigo contagiosa neonatorum.

2. The condition is benign and occurs within the first week of life. Apparently it is not transmitted by contact. All of our patients were Negroes.

3. The vesicle is the chief primary lesion. An occasional papule is sometimes seen.

4. The infant does not appear to be affected by the cutaneous infection. The temperature is practically normal throughout.

5. The condition may be complicated by a mild type of furunculosis. In no case was it necessary to resort to surgical measures to effect a cure.

6. In the only case in which bacteriologic examination was performed Staph. albus was the only organism isolated. A temporarily lowered resistance to this organism is postulated as an etiologic factor.

7. The points on which superficial pustular folliculitis of the new-born and impetigo contagiosa neonatorum may be differentiated are enumerated.

8. Superficial pustular folliculitis of the new-born responds promptly to baths with soap and water.

RECENT CHANGES IN DERMATOLOGIC DIAGNOSIS

RÉSUMÉ OF 1,112,050 PUBLISHED CASES

ANTON W. SOHRWEIDE, M.D.
NEW YORK

It is no exaggeration to say that in the continual progression of general medical knowledge in recent years dermatologic theory and practice have played no mean rôle.

Not only has there been a direct enlargement of the scope of dermatology brought about by the complex cutaneous reactions resulting from the constantly increasing flood of patented cosmetic and laxative preparations and from the introduction into industry of new chemical agents, but through valuable clinical research both here and abroad the therapeutic armamentarium has, in the aggregate, become finer and more accurate in recent years. For example, during the past five years, in private as well as in clinical practice, the gradual development of general knowledge concerning the practical importance of immune biologic processes as an aid in the diagnosis and logical classification of cutaneous diseases alone has revealed new vistas in dermatology—vistas that previously were unsuspected. The past five years of dermatologic progress have witnessed in a measure veritable exchanges of old lamps for new. The recent advances in the light of new facts and theories but herald future achievements; it is certain that the zenith has not been reached.

Statistical study of a large series of cutaneous diseases was undertaken in order to observe and to record possible changes in dermatologic diagnosis in the light of recent advances which have increased the cutaneous armamentarium since a similar report by Goodman five years ago.[1] In the present report important variations indicative of the new progress are recorded.

MATERIAL STUDIED

There was no selection of cases; the ten most common dermatologic diagnoses in the clinic of the Stuyvesant Square Hospital as recorded in résumé by the staff for the period from 1928 to 1933 are compared here with the published findings of previous investigators. Syphilis is purposely omitted, since previous reports do not afford a basis for

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From the New York Post-Graduate Medical School.

1. Goodman, H.: Statistics on the Ten Most Common Skin Diseases: Based on an Analysis of 973,090 Published Cases, Arch. Dermat. & Syph. **20**:186 (Aug.) 1929.

comparative study. To avoid confusion, each of the recorded diagnoses is interpreted in its fullest degree, so that tinea, for example, embraces all varieties such as cruris, circinata, versicolor, dyshidrosis and pompholyx; acne includes rosacea, indurata, vulgaris and medicamentosa; verruca includes vulgaris, plantaris, palmaris and senilis.

The figures as they appear up to 1927 are taken for comparative study from Goodman's [1] compilation. The present statistics add 138,960 new cases to those already recorded by Goodman. It is in this new group that distinct departures from previous observations were found; the influence of new dermatologic knowledge has left its impress here.

TABLE 1.—*Compilation of Published Statistics on Order of Incidence of Most Common Diseases of the Skin*

Disease	Crocker	Bulkley	Pollitzer	Pollitzer	S. & C.	S. & C.	S. & C.	St. Sq.
Order of			679,376		44,346	62,698	108,283	138,960
Total of	10,000	10,000	A. D. A.	58,387	Clinic	Clinic	Clinic	Clinic
1,112,050	Public	Clinic	1878-1911	A. D. A.	1913-1917	1918, 1919	1923-1927	1928-1933
Patients*	Patients	Patients	Inclusive	1916	Inclusive	1920 and 1922	Inclusive	
Eczema	1	1	1	1	1	1	1	2
Acne	5	2	2	2	2	2	2	3
Scabies	3	5	3	5	6	6	3	7
Psoriasis	4	3	10	4	5	7	7	8
Seborrhea	—	9	5	6	4	4	4	5
Impetigo	2	10	4	3	9	3†	8	6
Urticaria	6	6	9	8	3	5	6	9
Dermatitis venenata	—	4	11	10	7	8	5	1
Tinea	—	8	8	9	8	9	9	4
Alopecia	—	—	6	11	—	—	—	10
Pediculosis	—	—	7	12	—	—	—	13
Pruritus	8	7	12	14	11	11	11	11
Verruca	—	11	13	13	10	10	10	7
Ecthyma	—	—	—	7	—	—	—	—

* There may be some reduplication, as members of the American Dermatological Association at the New York Skin and Cancer Hospital undoubtedly reported their hospital patients for 1916 to Dr. Pollitzer.

† Based on the years 1919, 1920 and 1922.

INTERPRETATIONS

It is graphically shown in table 1 that for the first time since the publication of similar figures by Crocker, as early as 1903, the unsatisfactory diagnosis of "eczema" has been superseded by a somewhat more specific diagnosis—dermatitis venenata. The increased general knowledge of immunologic reactions in dermatology has altered previous figures considerably, so that the diagnosis of tinea has tripled during the past five years largely at the expense of "eczema," and this diagnosis has taken fourth place instead of ninth, as in previous reported cases. The other six ranking diagnoses have undergone no great variation from those previously quoted. It is interesting to note, however, that the incidence of scabies, probably in response to the campaign for cleanliness in many public schools, has declined almost one third; certainly a for-

ward step has been taken in the eradication of this infection when compared with the figures of Hebra, who in 1843 reported that 2,197 of 2,723 cases in the Vienna General Hospital were cases of scabies.[2] From the data submitted in table 2, one is impressed with the reduction in the incidence of psoriasis; the incidence of acne and impetigo, however, has remained unchanged in the course of years.

TABLE 2.—*Compilation of Published Statistics of Percentage of Incidence of Most Common Diseases of the Skin*

Disease	Crocker	Bulkley	Pollitzer	Pollitzer	S. & C.	S. & C.	S. & C.	St. Sq.
Order of			679,376		44,346	62,698	108,283	133,960
Total of	10,000	10,000	A. D. A.	58,387	Clinic	Clinic	Clinic	Clinic
1,112,050	Public	Clinic	1878-1911	A. D. A.	1913-1917	1918-1919	1923-1927	1928-1933
Diseases	Patients	Patients	Inclusive	1916	Inclusive	1920 and 1922	Inclusive	
Eczema	26.3	26.11	22.5	16.1	20	24.0	21.0	15.9
Acne	4.44	10.44	7.8	7.1	13	10.4	10.0	10.7
Scabies	7.96	2.13	4.9	3.2	4	5.0	5.0	3.5
Psoriasis	7.18	3.18	2.8	3.6	4	4.1	4.0	2.8
Seborrhea	....	1.12	3.9	3.1	4	5.1	5.0	5.02
Impetigo	9.61	0.99	3.8	4.3	2	5.5*	3.0	4.40
Urticaria	4.40	1.80	2.9	2.9	5	5.1	5.0	2.5
Dermatitis venenata	....	2.56	1.6	2.5	3	3.0	5.0	17.9
Tinea	3.18	0.96	3.2	2.8	3	2.0	2.0	10.2
Alopecia	....	....	3.0	2.1	..	...	...	1.8
Pediculosis	....	....	3.4	1.9	..	...	...	0.3
Pruritus	0.90	1.24	1.6	1.2	1	0.8	0.8	1.8
Verruca	....	1.14	1.0	1.6	1	1.5	1.0	2.9
Ecthyma	....	....	...	3.1	..	...	...	...

* Based on years 1919, 1920 and 1922.

The difference in statistics as to the six most common diseases of the skin is graphically shown in the following tabulation, together with a recent difference in the order of prevalence and a slight increase in percentage as compared with previously published figures.

		Percentage
Crocker:[3]	Eczema, impetigo, scabies, psoriasis, acne, urticaria	57
Bulkley:[4]	Eczema, acne, psoriasis, dermatitis venenata, scabies, urticaria	44
Pollitzer:[5]	Eczema, acne, scabies, impetigo, pediculosis	46
Pollitzer;[6]	Eczema, acne, impetigo, psoriasis, scabies, seborrhea	46
S. & C.:[7]	Eczema, acne, urticaria, seborrhea, psoriasis, scabies	50
S. & C.:[7]	Eczema, acne, impetigo, seborrhea, psoriasis, scabies	54
S. & C.:[7]	Eczema, acne, scabies, seborrhea, dermatitis venenata, urticaria	51
St. Sq.:[8]	Dermatitis venenata, eczema, acne, tinea, seborrhea, impetigo	64

2. Pusey, W. A.: History of Dermatology, Springfield, Ill., Charles C. Thomas, Publisher, 1933, p. 102.

3. Crocker, H. R.: Diseases of the Skin, Philadelphia, P. Blakiston's Son & Co., 1903, p. 1401.

4. Bulkley, L. D.: Diseases of the Skin, New York, Paul B. Hoeber, Inc., 1898, p. 41.

5. Pollitzer, S.: J. Cutan. Dis. **32**:312 (April) 1914.

6. Pollitzer, S.: J. Cutan. Dis. **36**:294 (May) 1918.

7. New York Skin and Cancer Hospital Annual Reports.

8. Stuyvesant Square Hospital (formerly New York Skin and Cancer Hospital) Annual Reports.

As shown in the foregoing data, in general dermatologic practice the ranking six diagnoses account for 64 per cent of all diagnoses in over 100,000 patients, a slight increase over previous figures quoted by Crocker, Bulkley, Pollitzer and Goodman. These statistics offer much for conjecture; it is interesting to learn that statistical study of a large series of diagnoses made at a cutaneous clinic during the past five years has recorded distinct variations from those previously made at the same clinic, among the same class of patients of like economic status and living under the same environment. The significant finding is that despite many progressive steps taken in dermatology during the past years, particularly in furthering learning relative to the rarer eruptions, the list of ten common diseases of the skin is still the cutaneous Armageddon.

News and Comment

AMERICAN BOARD OF DERMATOLOGY AND SYPHILOLOGY

The next meeting of the American Board of Dermatology and Syphilology will be held at the time of the meeting of the Southern Medical Association in San Antonio, Texas, on Nov. 13 to 16, 1934, if there are a sufficient number of applicants for the examination to warrant the trip. The oral examination for applicants in group A and group B will be held at this time.

The written examination for group B applicants will be held on Oct. 1, 1934, in various centers throughout the country.

All applications for this examination should be filed in the office of the secretary before Aug. 20, 1934.

Application blanks and booklet of information may be obtained from the secretary, Dr. C. Guy Lane, 416 Marlborough Street, Boston, Mass.

Abstracts from Current Literature

EDITED BY DR. HERBERT RATTNER

ERYTHEMA NODOSUM AND TUBERCULOSIS. HAROLD ERNBERG, Am. J. Dis. Child. **46**:1280 (Dec.) 1933.

From clinical and roentgenologic observations in well over 200 cases of erythema nodosum, the author comes to the conclusion that erythema nodosum is to be regarded in general as a symptom complex of a tuberculous nature and that the tuberculous focus is in a great majority of cases in the region of the pulmonary glands and only rarely in some other region. Further, he regards the symptom complex of erythema nodosum as an autogenous reaction to tuberculin and thus a process of an anaphylactic or allergic nature.

ECZEMA OF INFANCY AND CHILDHOOD: I. CONTACTS AS ETIOLOGIC AGENTS, WITH PARTICULAR REFERENCE TO FEATHERS. SAMUEL M. PECK and GUSTAV SALOMON, Am. J. Dis. Child. **46**:1308 (Dec.) 1933.

Fifty cases of eczema in infancy and childhood were studied. The patients were tested with sixty-four substances by means of scratch and patch tests, and one hundred and fifty-seven positive reactions to the skin tests were obtained. Eighteen food proteins elicited sixty-eight positive reactions in scratch tests and seventeen positive reactions in patch tests. On the other hand, twenty-one contact substances in patch tests gave sixty-two positive reactions. "There was an inverse ratio of positive reactions to scratch and patch tests made with food proteins as compared with the ratio of such reactions to these tests made with contact substances (inhalants). Among the food proteins, egg-white and egg yolk evoked the greatest number of positive reactions. Among the contact substances, goose, chicken and duck feathers elicited the greatest number of positive reactions. Egg-white and egg yolk in scratch tests evoked twenty-five positive reactions and in patch tests only eight positive reactions. The feather group in patch tests evoked thirty-eight positive reactions and in scratch tests only three positive reactions."

Scratch tests, even when the results were positive, were of no value in tracing the etiologic factors in cases of eczema. "Positive reactions to scratch tests made with egg-white or egg yolk are usually only an index to allergy of the skin; they have no bearing on the etiology of the eczema. Neither the feeding nor the elimination of eggs in the majority of the fifty cases influenced the course of the eczema."

The authors conclude: "The pathogenesis of eczema in infancy and childhood does not differ from that of eczema in the adult. The results of our investigations lead us to define it as an allergic reaction of the skin which is brought about by the contact of the patient with a substance or substances to which the skin is sensitive. The sensitivity may be hereditary, but is usually acquired. The eczema is caused as a rule by contact with a substance from without. Less frequently the allergen may be brought to the skin hematogenously, as in the case of foods."

XANTHOMATOSIS (SCHÜLLER-CHRISTIAN SYNDROME): REPORT OF A CASE WITH NECROPSY. KATHERINE K. MERRITT and BERYL H. PAIGE, Am. J. Dis. Child. **46**:1368 (Dec.) 1933.

A case of generalized xanthomatosis (Schüller-Christian's syndrome) with characteristic skeletal changes, exophthalmos, diabetes insipidus and cutaneous lesions is reported. Of four patients admitted to the Babies' Hospital in New

York with this syndrome, the first had a dry skin, the second showed xanthomas of the skin after discharge from the hospital and the third showed no cutaneous lesions. The fourth, whose case is reported, had cutaneous lesions of a papulopustular hemorrhagic nature on the trunk. These lesions evidently were xanthomas; at least, xanthoma-like cells were present in histologic sections. The cutaneous changes reported in association with other similar cases in the literature are briefly described.

So-Called Thymic Hyperplasia: IV. A Follow-Up Study of Thirty Cases. George W. Waldbott and G. E. Anthony, Am. J. Dis. Child. **47**:34 (Jan.) 1934.

The authors found in a study of the pathology of so-called thymic death a pulmonary edema which could not be explained on the basis of cardiac or renal disease and which was suggestive of an allergic origin. They therefore examined thirty patients in whom previous roentgenograms had revealed an enlarged thymus. These patients were studied for the purpose of discovering any possible association with allergy. They found that twenty-four of the thirty patients were afflicted with an allergic disease, as evidenced by the history and by cutaneous tests. In one of the patients a marked sensitivity to walnut was encountered, so severe that a scratch test with walnut produced a definite anaphylactic reaction. The possible relationship of thymic enlargement to anaphylactic death is discussed.

Anderson, Los Angeles.

Tularemia Treated by a New Specific Antiserum. Lee Foshay, Am. J. M. Sc. **187**:235 (Feb.) 1934.

The serum used in this work was obtained from a vaccinated goat, the method of production to be reported later. The best results were obtained when the serum was administered early in the course of the disease, though good effects followed its use as late as the second or third month. Good results are to be expected unless the liver and spleen have become infiltrated.

Sensitization tests after cure indicated that the serum cured by a desensitizing process: The more rapid and complete the desensitization, the more rapid the cure.

In vaccine-treated patients symptoms of the disease disappeared with the establishment of desensitization. In untreated patients a state of hypersensitivity persisted for at least fifteen months.

All experience indicates that the method is curative by a desensitizing process but not prophylactic.

Early diagnosis and treatment tend to diminish the incidence of suppurative adenitis, and a comparison of treated and untreated patients shows a 50 per cent reduction in the duration of the disease in general.

Warning is given not to use heat-killed antigens in making intradermal tests, as serious consequences may result.

A Hemorrhagic Eruption of the Mouth and Throat in the Rheumatic State. E. Holtz and G. Friedman, Am. J. M. Sc. **187**:359 (March) 1934.

The writers describe a transitory, deep red eruption which occurs and recurs on the mucous membrane of the mouth and throat, chiefly in cases of chronic rheumatic endocarditis.

The lesions are small, circular and nonelevated; one or several may be present; they are most frequently seen on the buccal mucosa and vary from the size of a pinpoint to 2 mm. in diameter. They vary in color from the crimson of fresh hemorrhage but may be darker or bluish. The duration is brief, the lesions often disappearing or fading considerably in twenty-four hours and being succeeded by a new crop.

The authors regard the eruption as a nonembolic, hemorrhagic enanthem.

Jamieson, Detroit.

SYPHILITIC ALBUMINURIA. P. H. WOSIKA and F. M. THURMON, Am. J. Syph. **18**:2 (Jan.) 1934.

Three cases of albuminuria apparently of syphilitic origin are reported and discussed. The interesting feature of these cases is that the evidence of renal impairment did not disappear until after from many months to several years of antisyphilitic therapy.

TREATMENT OF NEUROSYPHILIS WITH ACETARSONE (STOVARSOL) GIVEN INTRAVENOUSLY. L. SPIEGEL, Am. J. Syph. **18**:56 (Jan.) 1934.

A sodium acetarsone solution was prepared by dissolving 1 Gm. of acetarsone in 9 cc. of normal sodium hydroxide. To that was added 11 cc. of sterile distilled water. After filtering, the solution was ready to be administered intravenously. Twenty-five patients were treated with this preparation over a period of about seventeen months. An injection of 1 Gm. was given either weekly or twice weekly. There were no rest periods.

Judged by symptomatic and serologic improvement, the preparation appears to be efficient and rapid in producing results in the treatment of the various manifestations of neurosyphilis. No intolerance to the drug developed over long periods of time, and there was relative freedom from reactions.

THE SIGNIFICANCE OF THE AMERICAN DISTRIBUTION OF TABES AND PARESIS. W. F. PETERSEN, Am. J. Syph. **18**:75 (Jan.) 1934.

By means of a series of charts, the author points out the striking concordance between the track of tabes and dementia paralytica in America with the major storm tracks as they cross the continent. It seems warranted to associate at least two factors in the etiology of these neurosyphilitic conditions: first, meteorologically induced periods of vascular spasm that become apparent in the organs with terminal vascularization (central nervous system, heart, walls of the larger vessels, etc.), and second, an infection with Spirochaeta pallida. In this combination one deals with tissue degeneration induced by anoxemia and with inflammatory reactions partly due to anoxemic stimuli and partly due to infection. Other environmental factors besides meteorologic ones will also bring about periods of vascular spasm, and to the emotional trauma associated with the highly competitive existence in modern civilization one may with justice ascribe some of the alterations.

REUTER, Milwaukee.

CALCIFICATION OF THE SKIN IN DIABETES MELLITUS. A. H. DAVIS and S. WARREN, Arch. Path. **16**:852 (Dec.) 1933.

Two cases are reported of elderly diabetic women in whom calcification occurred in the blood vessels and as spicules of bone in the corium and subcutaneous tissue. The clinical course and the results of chemical examination of the blood in each case did not differ from those usually seen in diabetes mellitus, and the patients did not receive viosterol. There was no evidence of any abnormality of the parathyroid glands or of any of the usual causes for metastatic calcification.

CARO, Chicago.

LATE CONGENITAL SYPHILIS: A STUDY OF THE RESULTS OF TREATMENT IN 267 PATIENTS. FRANK R. SMITH JR., Bull. Johns Hopkins Hosp. **53**:231 (Nov.) 1933.

Smith studied the results of treatment of 267 patients suffering from congenital syphilis. All of these patients had been observed for more than two years. His data indicate that the "clinical arrest of late congenital syphilis, with or without obvious lesions, unless the nervous system is involved, may be accomplished with as little as 6 months of antisyphilitic treatment with an arsphenamine and a heavy metal; that the clinical result is the same regardless of whether the

Wassermann becomes negative or not; and that the optimal amount of treatment depends largely on the disappearance of clinical signs and symptoms, varying between 6 months and 3 years. The response of the blood Wassermann may be completely disregarded as a guide to the type of treatment or to its duration."

THE PASSAGE OF ARSENIC THROUGH THE HUMAN PLACENTA FOLLOWING ARSPHENAMINE THERAPY. N. J. EASHMAN and A. L. DIPPEL, Bull. Johns Hopkins Hosp. **53**:288 (Nov.) 1933.

This important contribution attempts to solve the following questions: Does the human placenta following arsphenamine therapy transmit the drug to the fetus? If so, what proportion of the injected arsphenamine is received by the fetus? Is the process one of simple diffusion so that the concentration of arsenic in the fetal blood at all times approximates that in the maternal blood? Or is some other mechanism involved?

These questions are answered as follows:

1. Following the intravenous administration of arsphenamine and neoarsphenamine to pregnant women, no arsenic is demonstrable in the fetal blood, although large quantities may be present in the maternal circulation.

2. Arsenic does pass to the fetus and is recoverable from meconium in substantial concentration. In the meconium of new-born infants, arsenic is constantly present when the mothers have had arsphenamine therapy during pregnancy.

3. Following arsphenamine treatment during pregnancy arsenic does not pass from the mother to the fetus by a process of simple diffusion but is transmitted in minute quantities by some other mechanism.

EXPERIMENTAL YAWS: II. COMPARISON OF THE INFECTION WITH EXPERIMENTAL SYPHILIS. THOMAS B. TURNER and ALAN M. CHESNEY, Bull. Johns Hopkins Hosp. **54**:174 (March) 1934.

In an attempt to decide the question of the identity or nonidentity of yaws and syphilis, these investigators obtained spirochetes from cases of both yaws and syphilis encountered in the same locality. They then inoculated rabbits to determine whether there were any differences in the behavior of the two diseases. Their results were as follows:

No differences were noted in the experimental infection in rabbits inoculated with the eight strains of yaws virus.

The disease picture produced by the yaws virus presented striking and for the most part constant differences from that produced by the Haiti strain of syphilis.

The Haiti strain of syphilis gave rise to an experimental disease in rabbits which was similar in every way to that produced by several strains of syphilis virus isolated in the temperature zone.

ANDERSON, Los Angeles.

SYPHILIS AND PREGNANCY. J. L. MCKELVEY and T. B. TURNER, J. A. M. A. **102**:503 (Feb. 17) 1934.

The authors report on a series of 943 pregnancies occurring in syphilitic women. As aids in establishing the diagnosis of congenital syphilis in the infant, the Wassermann test on the blood of the umbilical cord, histologic examination of the placenta and roentgen examination of the epiphyses of the long bones were most valuable. The percentage of fetal deaths and of syphilitic infants was greatly reduced by antepartum treatment, the good results being proportional to the amount of treatment given and to the time at which it was begun.

CARO, Chicago.

PHASES OF THE METABOLISM OF TRICHOPHYTON INTERDIGITALE PRIESTLY. DAVID R. GODDARD, J. Infect. Dis. **54**:150 (March-April) 1934.

This paper is a study of certain phases of the physiology of Trichophyton interdigitale Priestly and also includes some work on Microsporon lanosum Sabouraud. It is the result of evident painstaking effort but is not apt to be of as much immediate moment to the practical dermatologist as to the mycologist.

MICROSCOPIC DEMONSTRATION OF ACID-FAST BACILLI IN TUBERCULOUS FILTRATES AND THE PRODUCTION OF TUBERCULOUS "ULTRAVIRUS" INFECTIONS IN GUINEA-PIGS. ERNEST LINWOOD WALKER and MARION A. SWEENEY, J. Infect. Dis. **54**:182 (March-April) 1934.

This study was a repetition of the work and an examination of the claims of a number of investigators that there is an ultravirus stage of the tubercle bacillus in which it is capable of infecting the guinea-pig. They substantiated the claims of Fontes, Vaudremer, Calmette and many others that an atypical infection of guinea-pigs may result from inoculation with filtrates of tuberculous material through supposedly bacteria-proof filters. They were able to demonstrate the frequent passage of a few acid-fast bacilli through all types and grades of filters used by these investigators by microscopic examination of stained smears of centrifuge precipitates of the filtrates. Atypical nonprogressive infections ascribed to the "ultravirus" were produced by inoculation with tuberculous filtrates which were proved microscopically to contain a few tubercle bacilli. The authors assert that the frequent passage of a few tubercle bacilli through bacterial filters accounts for all of the phenomena consequent to inoculation with tuberculous filtrates without invoking the existence of Calmette's tuberculous ultravirus. They believe that the microscopic demonstration of the passage of tubercle bacilli through supposedly bacteria-proof filters creates presumptive evidence that other bacteria may also pass filters, evidence that will have to be taken into consideration in every claim for filtrable forms of other micro-organisms.

[This work is of interest to dermatologists because the authors seem to have found a flaw in the work of the French school which has given rise to the hypothesis that tuberculids are caused by the tubercle bacillus in the granule stage, tuberculosis granulimée.]

THE FILTRABILITY OF THE ACID-FAST GROUP. FRANK B. COOPER, J. Infect. Dis. **54**:236 (March-April) 1934.

The author was unable to confirm the work of those who claimed to have found a filtrable stage of the tubercle bacillus. CORNBLEET, Chicago.

LYMPHOGRANULOMA INGUINALE. MAX S. WIEN and M. O. PERLSTEIN, M. Rec. **139**:288 (March 21) 1934.

The various clinical phases of lymphogranuloma inguinale occurring in five American-born Negro women are reported, together with the results of necropsy in one case. The clinical sequence of the disease as it occurs in the female is described, varying from esthiomene, which follows an involvement of the superficial inguinal lymphatics, to the genitorectal syndrome, which follows involvement of the deep perirectal lymphatics and which proceeds to periproctitis, rectal stricture and rectovaginal fistulas. In the one case in which necropsy was performed, ulcerative proctitis and periproctitis with perirectal abscess formation, granular colpitis and a rectovaginal fistula were found, giving pathologic confirmation to the clinical sequence described. Four of the patients had repeated positive Frei tests. The occurrence of a negative Frei test in the case of the fifth patient was interpreted as due to a state of anergy. The clinical diagnosis of lymphogranuloma inguinale in this case was confirmed by cross-testing and by biopsy on tissue from the involved buttocks. AUTHOR'S ABSTRACT.

PERSONAL IMPRESSIONS IN THE TREATMENT OF A FEW OF THE COMMONER INVETERATE DISEASES OF THE SKIN. FRED WISE, New York State J. Med. **33**:1321 (Nov. 15) 1933.

One of the old remedies, arsenic, has in Wise's experience proved its worth beyond question in the treatment of chronic psoriasis, eczema, urticaria, acne and selected cases of neurodermatitis. He recommends the subcutaneous injection of a 2 to 4 per cent solution of sodium arsenate given in quantities approximating the maximum tolerated dose; if injections are not feasible arsenic in the form of pills should be employed. The value of the roentgen rays and of a solution of sulphurated lime in acne and sycosis is discussed, as well as the value of the roentgen rays and of Dreuw's ointment in psoriasis and neurodermatitis. The use of ordinary vaccination for recurrent herpes simplex is highly recommended, and the question of desensitization by means of trichophytin in tinea is touched on.

TRAUB, New York.

CUTANEOUS ONCHOCERCIASIS IN A SIMULIUM DAMNOSUM-INFESTED REGION IN UGANDA. E. G. GIBBINS and L. J. A. LOEWENTHAL, Ann. Trop. Med. **27**:489 (Dec. 20) 1933.

In the cases discussed, all stages of the disease were presented, from a papular or papulopustular eruption to a general lichenification or pachydermia.

A routine examination for subcutaneous nodules resulted in finding the nodules in one third of the cases of lichenification. Adult nematodes were found in the nodules on dissection.

Other cases of lichenification had been seen in which the thickening of the skin apparently followed other dermatoses, but there were no subcutaneous nodules, the cases were not found in Simulium damnosum-infested regions and larvae were not found on biopsy.

JAMIESON, Detroit.

PAPULAR URTICARIA AND THE DYNAMICS OF SKIN SENSATION. D. W. WINNICOTT, Brit. J. Child. Dis. **31**:5 (Jan-March) 1934.

The pathogenesis of papular urticaria is dependent on two factors: First, the skin is normally an excitable organ, and papular urticaria is an expression of this excitement, corresponding to excitement of erectile tissue in other parts of the body.

The complicated, distressing form of papular urticaria is usually due to the second factor, namely, a complicated psychologic disorder "involving some degree of repression in connection with more advanced auto-erotic gratification." This type of papular urticaria is very frequent, especially with changing emotional states, or may be brought about by external causes, such as unsuitable clothing, preexisting dermatoses or internal causes which lead to various degrees of sexual excitement associated usually with masturbation.

Other pruritic cutaneous conditions which may have a psychologic basis are pruritus ani, chilblain and infantile eczema, in all of which pleasure is usually derived from friction.

PERLSTEIN, Chicago.

PROGRESSIVE ULCERATIVE RETICULOSIS OF THE PALATE. I. S. HALL, J. Laryng. & Otol. **49**:35 (Jan.) 1934.

Hall describes the case of a woman, aged 32, with a progressive ulcer of the palate. The ulcer possessed no definite characteristics. Biopsy revealed vesiculation and edema and aggregations of cells showing mitosis and evidence of active growth. After a rapid febrile course, during which bullae developed on the bridge of the nose, in the groin and over the thorax, the patient died. On postmortem examination, in addition to the local and cutaneous lesions septic leptomeningitis with extreme peripheral congestion and hemorrhages into the subarachnoid cavity were found. The infection had extended from the sphenoid bone through a por-

tion of the lamina cribrosa, which was necrotic. There were pulmonary congestion and edema, and within the parenchyma there were three or four small firm, white nodules. Similar nodules occupied the cortex of both kidneys. Histologic investigations of the nodules in the kidneys and lungs showed features similar to those in the epidermis, namely, aggregations of cells of various sizes undergoing mitosis. The author believes that death was due not directly to sloughing, hemorrhage or toxemia but to the leptomeningitis, an almost accidental complication. He reviews thirteen similar cases found in the literature. He suggests that the specific cells which were so prominent a feature of the initial ulcer and of the subsequent lesions may have originated in the reticulum cells of the lymphoid tissue of the palate. Such a conception links this ulceration with agranulocytosis, with diseases of the bone marrow and of the stroma and with lymphogranuloma. Moreover, it cannot be overlooked that the areas first affected are apt to be those which are normally the site of copious micro-organismal colonization.

EDITOR'S ABSTRACT. [ARCH. OTOLARYNG.]

STUDIES IN DERMAL LEISHMANOID: PART I. P. BRAMACHARI, J. Trop. Med. **37**:68 (March 1) 1934.

A rare type of dermal leishmanoid is described, occurring in two brothers who presented the same type of lesions. The symptoms consisted of well marked areas of hyperpigmentation, extensive areas of depigmentation, a small number of papules on the body, localized erythema and the absence of nodules in the skin.

JAMIESON, Detroit.

OCULAR COMPLICATIONS OF ACNE ROSACEA. B. ADAMENTIADIS, Ann. d'ocul. **170**:760 (Sept.) 1933.

The effect of acne rosacea of the face on the eye, particularly on the cornea, was first described by Arlt in 1864. Since that time there have been many communications by different authors, including the complete study of Peters and his pupils Erdmann and Triebenstein, who determined the different forms in which this malady shows itself in the eye.

Among 5,000 patients Adamentiadis saw 3 with ocular rosacea, and on the basis of the material in the ordinary textbooks he concludes that the condition is rare. One of its principal features is the telangiectasia of the already congested coats. This is found usually in the locality of the acne rosacea, on the conjunctiva and the sclera and part of the cornea. It is localized and of an eruptive nature. The veins appear dilated and tortuous around the acne lesion. Another feature of the malady, equally important, is the chronicity, which is discouraging both to the patient and to the physician.

In the first two of the three cases reported there was nothing of special interest. In the third case, however, the patient, aged 7 years, had had the lesion since his first year. The lesions of the anterior parts of the eye had healed and recurred constantly, taking a chronic course slightly different from that usually seen.

MCKEE, Montreal, Canada. [ARCH. OPHTH.]

CHEMICAL METHOD OF CORNEAL TATTOOING FOR PRODUCTION OF A BROWN COLOR. F. SPINELLI, Arch. di ottal. **40**:266 (June) 1933.

The author reviews the literature on chemical staining of the cornea, most of which is devoted to imitating the black pupil. Since in staining with gold chloride the intensity of color depends on the activity of the reducing agent employed, the author searched for a reducing agent weaker than hydrazine hydrate while of the same general properties. This he found in hydrazine hydrochloride. By varying the strength of the gold chloride and hydrazine hydrochloride solutions in the test tube, a range of colors could be obtained from dark brown with 4 per cent gold chloride and 4 per cent hydrazine hydrochloride to light brown,

resembling the shade of a hazel iris, with 4 per cent gold chloride and 2 per cent hydrazine hydrochloride. Still lighter tones were produced by further dilution of the reducing agent.

In a group of rabbits the right eyes were stained by the second method and the left eyes by the first method, the gold solution being applied for two minutes after removal of the central corneal epithelium and the reducing agent for one minute. A uniform light brown stain was produced with the more dilute reducing agent, and a darker brown with the more concentrated solution. The epithelium formed over the stained area in from three to six days, and the reactions were minimal.

Sections of eyes removed after one, three, seven, twenty and thirty-five days showed that the fine granular precipitate of brown particles occurs first in Bowman's membrane and then migrates farther back into the corneal stroma. The granules in the more posterior layers become clumped in larger particles. There was only a slight infiltration of the deeper portions of the cornea with eosinophil cells which contained brown granules.

The method was employed in two cases of corneal scars in patients with brown irides. In the first case the central portion of the cornea had been stained with platinum chloride and hydrazine hydrate. The surrounding portion of the cornea was stained with 4 per cent gold chloride and 2 per cent hydrazine hydrochloride five months later, and five months after this operation a shiny brown color was present which contrasted well with the black central stain.

In the second case a scar in the lower quadrant was stained with 4 per cent gold chloride and 2 per cent hydrazine hydrochloride. Ten months later the brown color remained unchanged and was almost identical with that of the healthy eye.

GIFFORD, Chicago. [ARCH. OPHTH.]

REPORT OF A SYPHILITIC DISEASE OF THE CONJUNCTIVA. F. VON PAPOLCZY, Arch. f. Augenh. **108**:334, 1933.

In a previous communication (*Arch. f. Augenh.* **103**:323, 1930) von Papolczy described a diffuse thickening of the conjunctiva in a 20 year old girl who had a strongly positive Wassermann reaction. The condition cleared up rapidly under appropriate antisyphilitic treatment and was therefore believed to be syphilitic.

In the present article two conditions are described which were quite unlike in their clinical appearance, but which occurred in patients with strongly positive Wassermann reactions and which disappeared under antisyphilitic treatment.

A 39 year old man who had acquired syphilis four months previously showed a uniform thickening of the bulbar conjunctiva, which invaded the limbus at the upper pole. The epithelium was intact, and the mass had a gelatinous appearance and was richly vascularized.

A 61 year old man who had acquired syphilis twenty-three years previously, but who still had a positive Wassermann reaction, presented a bilateral swelling of the lower lids, with thickening so marked that there was definite ectropion. The tissue of the lids was extremely hard. Excision of some of this tissue showed nothing pathognomonic of syphilis.

In both cases the lesions healed rapidly after intensive antisyphilitic therapy.

ADLER, Philadelphia. [ARCH. OPHTH.]

RHINOSCLEROMA AT NECROPSY. W. NOWICKI, Beitr. z. path. Anat. u. z. allg. Path. **91**:457, 1933.

Although rhinoscleroma is a common disease in the portion of Poland in which Lemberg is situated, few persons with this disease are seen on the necropsy table, evidently because the disease is rarely the immediate cause of death or of a terminal illness that leads to hospitalization. In Nowicki's institute at Lemberg 23 cases of rhinoscleroma were encountered within thirty-six years in a total of 31,962 necropsies. The disease may be the cause of sudden death through

asphyxia or through hemorrhage complicating tracheotomy. Nowicki included in his series 8 cases of this type from the local medicolegal institute. Males and females were equally affected. Most of the cases occurred in young persons; 5 occurred in the age period from 11 to 20 years, and 17 in the decade from 21 to 30 years. The process was not limited to any particular portion of the upper respiratory tract. The larynx was most often involved, but usually in association with the nose, trachea and primary bronchi. Four anatomic forms are described; nodose, tuberous, diffuse and cicatricial. The essential features of the 31 cases are presented in tabular form.

SCHULTZ, Evanston, Ill. [ARCH. PATH.]

CUTANEOUS CHANGES RESULTING FROM LIGHTNING. PAUL FASAL, Dermat. Ztschr. **68**:105 (Dec.) 1933.

Three patients are described in whom branched figures occurred on the skin as a result of their being struck by lightning. Histologic examination of a biopsy specimen removed six hours after the accident revealed intra-epithelial vesicles, disturbance of the intra-epithelial bridges in the stratum spinosum and a perivascular infiltrate of small round cells and neutrophilic and eosinophilic leukocytes.

NAEVUS MULTIPLEX PRINGLE AND MORBUS RECKLINGHAUSEN. THEODERICH NIMPFER, Dermat. Ztschr. **68**:112 (Dec.) 1933.

A patient is described who had adenoma sebaceum. In addition, he presented neurofibromatosis and a van der Hoeve tumor of the right eye. This combination speaks for the close relationship of these forms of maldevelopment.

DERMATOFIBROMA LENTICULARE (SCHREUS). KLÄRE GÜLDEN, Dermat. Ztschr. **68**:118 (Dec.) 1933.

As a result of a histologic and clinical study of thirteen patients with lenticular dermatofibroma, the author concludes that these lesions clinically resemble keloids most closely but histologically are nearer to fibromas.

DEMONSTRATION OF TUBERCLE BACILLI IN THE CIRCULATING BLOOD BY CULTURE AND ANIMAL INOCULATIONS. FRANZ KOCH, Dermat. Ztschr. **68**:127 (Dec.) 1933.

The author was unable to demonstrate tubercle bacilli in the blood of patients with various types of tuberculosis either by means of animal inoculation or by the culture method of Löwenstein, whose technic was followed in every detail.

TAUSSIG, San Francisco.

HISTOPATHOLOGY OF SCLEROMA. B. L. FELDMANN, Monatschr. f. Ohrenh. **67**:1423 (Dec.) 1933.

Feldmann found that the classic characteristics of rhinoscleroma (Mikulicz' cells, bacteria and hyalin globules) are not always present and that their absence does not exclude a diagnosis of scleroma. Frisch's bacillus is often not found. The microscopic observation of granular, inflammatory tissue rich in blood vessels and containing numerous plasma cells and histiocytes, accompanied by obliteration of the vessels and hyalinization of the peripheral layers, is indicative of scleroma. This histologic picture, with a positive Bordet-Gengou reaction and an atrophic condition of the mucous membrane of the upper respiratory tract, may be considered characteristic of rhinoscleroma.

EDITOR'S ABSTRACT. [ARCH. OTOLARYNG.]

INTESTINAL RHINOSCLEROMA. W. ROTTER and PEÑA CHAVARRÍA, VIRCHOWS Arch. f. path. Anat. **289**:345, 1933.

Involvement of the alimentary tract in rhinoscleroma has not been hitherto described. In a case of the disease which came to necropsy in Costa Rica the mucosa of the cecum and ascending colon from the ileocecal valve to the hepatic flexure was studded with nodular elevations. The nodules were composed of granulation tissue that contained Mikulicz' cells and was histologically identical with the tissue of the nasal and upper respiratory tract lesions of rhinoscleroma. From their experience with this disease the authors have found that the number of Mikulicz' cells may vary greatly. The cells are most numerous in the recent and most acute lesions. Sometimes the histologic diagnosis must be based on the presence of hyaline cells. When the histologic changes are not characteristic, the diagnosis must depend on the isolation of the specific encapsulated bacillus.

SCHULTZ, Evanston, Ill. [ARCH. PATH.]

PSORIASIS OF TYMPANIC MEMBRANE. H. LEICHER, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:393, 1933.

Leicher describes three cases of psoriasis of the tympanic membrane, with perforation in two, which resulted from an injury caused by caustic or by radioactive salves. Treatment is slow and difficult, and there is a great tendency toward relapse, as in psoriasis of other parts of the body.

SIMULTANEOUS OCCURRENCE OF TUBERCULOSIS AND SYPHILIS. J. BECK, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:472, 1933.

Beck describes three cases of apparently simultaneous development of tuberculosis and syphilis. In the first, that of a child, aged 13, biopsy on a firm infiltration of the tongue showed epithelioid cell tubercles with giant cells, so that from a pathologic standpoint tuberculosis was diagnosed. After about nine months' treatment, in which the severe pain improved, the patient suddenly became hoarse, and the larynx showed deep infiltration. The Wassermann reaction at that time was 3 plus. Treatment with potassium iodide caused rapid improvement of the infiltration in the larynx and even some improvement of that of the tongue. In the other two cases there was evidence of the presence of both tuberculosis and syphilis. The author feels that the diagnosis of a mixed infection is often exceedingly difficult. There is some evidence that syphilis at times predisposes to tuberculosis. The histologic picture of syphilitic granulomas is often indistinguishable from that of the tuberculous lesions. A positive Wassermann reaction does not prove that a lesion is syphilitic, and a negative reaction does not rule out syphilis. In general, a tuberculous lesion is likely to be more painful than a syphilitic lesion.

EDITOR'S ABSTRACT. [ARCH. OTOLARYNG.[

GUMMA OF THE LOWER LID. RYSS-ZALKIND, Sovet. vestnik oftal. **1**:55, 1933.

A patient, aged 21, revealed on examination a firm swelling the size of a walnut at the inner angle of the eye. In two days the swelling broke down. The necrotic matter obtained from it was examined for spirochetes and tubercle bacilli, but none were found. Histologic examination showed connective tissue in a state of incomplete necrosis; in the walls of the blood vessels there were marked necrotic changes. The Wassermann reaction was positive. An ulcer with undermined, infiltrated edges and a deep fatty base appeared in the place of the former swelling. After five injections of neoarsphenamine the ulcer healed, and a fine cicatrix formed.

The diagnosis of gumma was made because of the appearance of the ulcer, the histologic observations, the absence of swelling of the regional glands and the absence of spirochetes in the lesion.

SITCHEVSKA, New York. [ARCH. OPHTH.]

SWEAT GLAND FUNCTIONING IN THE SPOTS OF LEPROSY. YASUNORI YOSHIMURA, Jap. J. Dermat. & Urol. **35**:105 (Feb.) 1934.

In about 100 patients with leprosy, diseased areas in various stages of development and of activity were tested for the normal loss of water, or perspiratio insensibilis, and then while in the sweating state. Sweating was induced by increased environmental temperatures and by injections of pilocarpine.

On the general body surface (exclusive of the palms and soles) the loss of water seems to be, in the nonsweating state, chiefly that of transpiration, the sweat glands apparently not taking part in the process. This conclusion is deduced from the fact that in perspiratio insensibilis leprous areas, where the infiltration and inflammatory reaction are not pronounced, do not show a loss of water much different from that of the rest of the body. This conclusion, however, does not apply in the case of the skin of the palms and soles, since here there is a noticeable diminution of loss of water in the diseased areas as compared with the remaining skin (postulating pathologic inhibition of the sweat glands in these macules).

In the sweating state the actively hyperemic areas show an increase in loss of water over that of the normal skin areas. However, in the macules, where there is no longer evidence of inflammatory activity (secondary leukoderma), there is a decrease in the amount of sweat given off, and in those of these areas that are markedly infiltrated there is at times actual anhidrosis. That this anhidrosis is not purely neurogenic is shown by the fact that when the infiltration is diminished in these areas a moderate amount of sweating reappears.

TUBERCULIN-ALLERGY AND TUBERCULOSIS OF THE SKIN. S. KITAMURA and K. TASAKI, Jap. J. Dermat. & Urol. **35**:157 (Feb.) 1934.

In histologic studies of specimens of skin excised from areas of reaction to patch tests and to the Mantoux test it was found that the manifestations in the epidermis are always accompanied by characteristic changes in and about the capillaries of the subcutis. Therefore, the conclusion that in tuberculosis of the skin the primary changes are in the vessels of the subcutis and the epidermal manifestations are secondary seems justified. The allergic disposition is primarily of the cutis and not of the epidermis.

This cutaneovascular sensitivity was reduced by the Gerson (salt-free) diet and inunctions of cod liver oil, and the results seem to indicate that this alteration of tissue disposition is of value in the treatment of cutaneous tuberculosis.

BRENNAN, Chicago.

SPIROCHETES IN THE LYMPH GLANDS OF SYPHILITIC RABBITS: PAPER I. S. WATANABE, Lues: Bull. soc. japon. de syph. **10**:1 (Oct.) 1933.

In order to investigate the spirochete content of the lymph glands of rabbits which had been infected with syphilitic virus Watanabe removed the lymph glands from six to three hundred and fifteen days following the infection, emulsified them and injected the emulsion in different concentrations into the backs of healthy rabbits. The author has found that the number of spirochetes in those glands varies considerably in the course of the syphilitic illness.

SPIROCHETES IN THE SPLEEN AND LYMPH GLANDS OF SYPHILITIC RABBITS AFTER INSUFFICIENT(?) TREATMENT WITH SALVARSAN. H. MISAIZU, Lues: Bull. Soc. japon. de syph. **10**:38 (Oct.) 1933.

Syphilitic rabbits received 38, 180, 509 and 622 days, respectively, after infection two injections of 0.03 Gm. of arsphenamine per kilogram of weight at an interval of 2 days. Twenty-one days later an emulsion of the spleen and lymph glands of each was injected in different dilutions into the skin of the back and under the skin of the scrotum of normal rabbits: No positive result was obtained.

The emulsion of spleen and lymph glands of rabbits which had been infected 101, 273 and 497 days previously when injected into normal rabbits gave positive results, while inoculation 786 days following infection gave a negative result.

A Case of Malignant Syphilis with Grave Involvement of the Mucous Membranes of the Mouth. S. Ninomiya, Lues: Bull. Soc. japon. de syph. **10**:50 (Oct.) 1933.

The author reports a case of secondary syphilis with an extensive eruption of miliary papules and pustules on the body. This eruption and lesions in the mouth, pharynx and larynx and on the conjunctivae resembled the picture of erythema multiforme. The patient showed serious involvement of the larynx, giving rise to difficult breathing and deglutition, which responded promptly to antisyphilitic treatment.

Bloom, New York.

Visceral Metastases of Cutaneous Metatypical Epitheliomas. M. Juon, Acta dermat.-venereol. **14**:287 (Nov.) 1933.

The author describes a case of epithelioma of the hand, the histologic structure of which was that of an intermediary metatypical epithelioma, described first by Darrier and Ferrand (the cells are neither basal nor squamous but bear a resemblance to both types). In the case described by the author the epithelioma gave rise to metastases, first to the lymph glands and then, through general hematogenous dissemination, to the lungs, liver, kidneys, small intestines, etc. Histologically, the metastases were also of the transitional cell type. The author believes that the intermediary metatypical epithelioma, though in most cases benign, may in certain instances give rise to metastases quite as malignant as those of squamous cell epitheliomas.

Comedo Nevus. Nelson Paul Anderson, Samuel Ayres Jr. and Louis M. Kane, Acta dermat.-venereol. **14**:299 (Nov.) 1933.

The authors describe two cases of comedo nevus, with the histologic observations in one of them. The literature is reviewed. In recent years authoritative investigators have included under the term "nevus" all disturbances of the skin present at birth or developing later in life from embryonic predisposing causes. On the basis of this fact and also taking into consideration the most important characteristics of the comedo nevus, the authors consider this condition a nevus. The differential diagnosis of comedo nevus, folliculitis ulerythematosa reticulata of MacKee and Parounagian, multiple benign cystic epithelioma, adenoma sebaceum and syringocystoma is discussed.

Further Contribution to the Question of Atrophy of the Skin: Acrodermatitis Chronica Atrophicans and Its Relation to Scleroderma. Eugenie Benjamowitsch and L. M. Maschkilleisson, Acta dermat.-venereol. **14**:313 (Nov.) 1933.

The authors describe twenty-seven cases of acrodermatitis chronica atrophicans (clinical and histologic picture). They distinguish three different stages: (1) inflammatory infiltrative, (2) atrophic and (3) scleroderma-like and tumorous. All three stages may be present in the same patient. Some cases were accompanied by trophic ulcers, while others presented osseous changes (thickening of the cortical layer on the anterior aspect of the tibia) in the scleroderma-like areas. While the precipitating cause may be a cold, trauma or infection, the predisposing factor is usually an endocrine disturbance, especially the disturbed functioning of the sexual glands (in women). While acrodermatitis atrophicans involves chiefly the elastic tissue, scleroderma is by its nature a disease principally of the collagenous tissue. The different methods of treatment of acrodermatitis atrophicans (diathermy, glandular therapy and forms) are discussed.

EPIDEMIOLOGY AND ETIOLOGY OF MILKERS' NODULES (MELKERKNOTEN). P. M. SALKAN, Acta dermat.-venereol. **14**:342 (Nov.) 1933.

The author saw milkers' nodules among eighteen milkmaids, the majority of whom presented lesions at the same time. This disease occurs mostly on the hands and fingers and is characterized by the presence of pea-sized, elevated, hard, sharply defined, painless, round lesions with central umbilication. The lesions do not ulcerate and heal without scarring. The cows which were milked by the milkmaids had simultaneously on their udders papular and pustular lesions (which were not cowpox) the histologic appearance of which was about the same as that of the milkers' nodules. The author is of the opinion that the condition is infectious and that the patients get it from diseased cows. The virus of milkers' nodules is biologically related to the smallpox virus; however, it is not identical with it, for (1) the immunity against smallpox does not protect against milkers' nodules and (2) the latter may recur after six weeks. According to the author, milkers' nodules are a paravaccinial disease the virus of which is different from the virus of the smallpox vaccine. Intracutaneous injection of the fluid withdrawn from milkers' nodules is of therapeutic value by shortening the course of the disease.

PURPURA ANNULARIS TELANGIECTODES (MAJOCCHI). GUNNAR NORDIN, Acta dermat.-venereol. **14**:369 (Nov.) 1933.

The author reports three cases observed in Sweden and cites the most important symptoms of the disease. He believes that because of the absence of any subjective and general symptoms Majocchi's disease is neglected by the patient and overlooked by the physician and therefore is seldom observed. He also discusses the histologic picture of the disease: dilatation of the papillary capillaries, perivascular cell infiltration, hemorrhages, hyaline degeneration of the vessel walls, endarteritis obliterans and atrophy. He gives a detailed description of a case which came under his own observation. ASTRACHAN, New York.

Society Transactions

DERMATOLOGICAL CONFERENCE OF THE MISSISSIPPI VALLEY

MAX S. WIEN, M.D., *Acting Secretary*

Seventh Annual Session, Chicago, Jan. 20, 1934

OLIVER S. ORMSBY, M.D., *in the Chair*

MALIGNANT ERYTHRODERMA; ALEUKEMIC LEUKEMIA (LYMPHATIC). Presented by DR. C. W. FINNERUD and DR. F. V. PLUMMER, Chicago.

This woman, was presented at the November 1933 meeting of the Chicago Dermatological Society, and the history was published in the transactions of that society (ARCH. DERMAT. & SYPH. **29**:766 [May] 1934).

LYMPHOBLASTOMA. Presented by DR. E. A. OLIVER, Chicago.

This patient was presented at the March 1932 meeting of the Chicago Dermatological Society, and the history is published in the transactions of that society (ARCH. DERMAT. & SYPH. **26**:1108, 1932).

At that time he showed generalized erythema which involved the entire body, with the exception of the face and neck. The skin was pinkish red and not infiltrated, and the color faded on pressure. The results of examination of the blood were within normal limits, but in the discussion the consensus was that the condition belonged to the lymphoblastoma group.

The patient was not seen again until Dec. 18, 1933, when the condition of the skin was the same as at the present time. He is a patient in the United States Veterans' Hospital. A fecal fistula which is present was the result of a gunshot wound in the abdomen. He states that his skin first took on a reddish hue in 1928, shortly after the gunshot wound, and has become progressively worse since that time.

On December 19, the leukocyte count was 17,300. The differential count showed: polymorphonuclears, 22 per cent; small mononuclears, 58 per cent; large mononuclears, 18 per cent, and eosinophils, 2 per cent. The urine was normal. Roentgen examination of the skull gave negative results. On Jan. 20, 1934, the erythrocytes numbered 4,250,000, and the leukocytes, 16,400. The differential count showed: polymorphonuclear neutrophils, 39 per cent; small mononuclears, 55 per cent; large mononuclears, 3 per cent, and eosinophils, 3 per cent.

Sections from biopsy specimens from the skin and from an inguinal gland are presented.

DISCUSSION

DR. UDO J. WILE, Ann Arbor, Mich.: I think that both of these cases are excellent examples of a rather vague picture in which much mutation can be seen from time to time and which represents either a primary change in the blood and bone marrow and secondary changes in the skin or a reaction of the skin to an insult with secondary changes in the blood. For a long time I have felt that it is difficult to give a name to such conditions other than malignant erythroderma, which perhaps is fitting on account of the malignancy of the condition. In some instances scaly erythroderma develops into leukemia, in some into granuloma fungoides, and in some into neither of these conditions but rather into Hodgkin's disease. Within the last month or two my associates and I have had a

strikingly brilliant example of the different clinical phases which such cases can pass through. In the case just presented, clinically no one would have hesitated to make a diagnosis of mycosis fungoides in 1932. The plaques were extremely pruritic, and we expected that the typical tumor stage of this disease would develop. The man returned this year with the clinical appearance of the skin the same but with the findings of Hodgkin's disease in the blood and lymph glands —an example of the fact that in such a case the picture can change from time to time either without treatment or without being influenced by treatment.

DR. LESTER M. WIEDER, Milwaukee: I think that both of these cases would be accepted clinically as instances of malignant erythroderma. The blood count is corroborative of the diagnosis in the second case, but in the first case the blood appears normal. In that case, however, the biopsy shows a dense infiltrate, particularly in the upper part of the cutis, predominantly lymphocytic, and I think that one must accept the lymphocytic infiltration of the dermis as a valuable diagnostic aid, especially when correlated with the fact that the patient also has enlarged lymph glands in all the superficial chains. This case well illustrates the fact that too much reliance must not be placed on the blood picture, and that biopsy is essential in all cases of suspected malignant erythroderma.

DR. PAUL A. O'LEARY, Rochester, Minn.: Another phase of the problem of lymphoblastoma which is of utmost importance is that of the pathologic changes in the cells. The character of the cellular infiltrate in the histopathologic sections in these diseases is of much significance and may be the only means of making a diagnosis. On the other hand, in a study of the blood smear the character of the cellular elements may be of more importance than the quantitative estimation of the cells; for example, the presence of stem cells in a blood smear may permit the diagnosis of leukemia before appreciable lymphocytosis has appeared. Accordingly, the aid of a hematocytologist must be sought for the early recognition of certain of the lymphoblastomas.

DR. C. W. FINNERUD, Chicago: I think that in our case the fact is well illustrated that almost from the beginning the histologic picture can be definitely that of the disorder which will later become apparent. The first biopsy specimen was taken a few months ago, and I think every one will agree that it definitely shows leukemia. Even though the condition has progressed to the present stage the blood is practically normal. The highest lymphocyte count has been about 25 per cent, both large and small, and the total number of leukocytes, only 14,000 per cubic millimeter. We can predict almost with certainty that the condition will terminate as true lymphatic leukemia.

DR. E. A. OLIVER, Chicago: When this man was first presented in 1932, at the age of 34, he stated that his skin had taken on the pinkish red color four years previously. There was no glandular adenopathy, the blood picture was within normal limits and the skin presented generalized erythema rather than erythroderma. At that time the diagnosis of lymphoblastoma was suggested by many of the members present.

I did not see the patient again until December 1933, when he presented the same symptoms that he shows today. The skin is deeper red, markedly infiltrated, desquamating in places and extremely pruritic. There is marked glandular adenopathy, and during the last month nodosities and infiltrations suggestive of those of granuloma fungoides have been developing.

He suffers intolerably from pruritus.

SCLERODERMA, GENERALIZED, WITH CALCIFICATION. Presented by DR. RUBEN NOMLAND and DR. W. W. TOBIN, Chicago.

This patient was presented at the November 1933 meeting of the Chicago Dermatological Society, and the history was published in the transactions of that society (ARCH. DERMAT. & SYPH. **29**:771 [May] 1934.

DISCUSSION

DR. PAUL A. O'LEARY, Rochester, Minn.: I raise the question as to whether this child has scleroderma or chronic polymyositis. The absence of true cutaneous sclerosis in the presence of a history of many years of incapacity is against a diagnosis of scleroderma. The fact that the greater part of the involvement seems subcutaneous rather than in the skin is also against such a diagnosis. Roentgenograms of the thighs and flanks might show the presence of calcium, a frequent complication of polymyositis. Scleredema and the residue of dermatomyositis must also be considered in the differential diagnosis but are readily eliminated.

DR. KARL G. ZWICK, Cincinnati: Recently I had the opportunity to examine an otherwise apparently healthy young man with scleroderma of the extremities. As his basal metabolism was found to be rather low, a thyroid preparation was administered. Improvement occurred for a time after, if not because of, this medication. Recently an extension of the sclerodermatous process has been noted in that patient. It would interest me to know whether a reduction of the metabolism has been recorded in a considerable series of cases of scleroderma.

DR. RUBEN NOMLAND, Chicago: In answer to Dr. O'Leary, there are calcium deposits in the muscles, and the diagnosis of generalized scleroderma was made with the idea that in scleroderma instead of sclerosis of the skin alone there probably is sclerosis of other structures, including the muscles. The acute onset of the sclerodermatous condition at the age of about 2 years seems incompatible with the diagnosis of myositis ossificans. I think that the child has a generalized sclerosing disorder with deposits of calcium, and so far as I can tell it should be called scleroderma unless some more satisfactory classification is brought forward.

PSEUDOXANTHOMA ELASTICUM OF THE SKIN; ANGIOID STREAKS OF THE RETINA; OLD CHORIORETINITIS. Presented by DR. RUBEN NOMLAND and DR. BERTHA KLIEN, Chicago.

This patient was presented at the October 1932 meeting of the Chicago Dermatological Society, and the history was published in the transactions of that society (ARCH. DERMAT. & SYPH. **27**:849 [May] 1933). The patient was also presented at the clinical meeting of the American Dermatological Association in June 1933, and a report was published in the transactions of that association (ARCH. DERMAT. & SYPH. **29**:147 [Jan.] 1934).

PSEUDOXANTHOMA ELASTICUM AND ASSOCIATED PURPURA WITH ANGIOID STREAKS. Presented by DR. O. H. FOERSTER, Milwaukee.

This man, aged 42, married, a typesetter for the past nineteen years, sought medical advice on Nov. 22, 1933, at the State of Wisconsin General Hospital, for purpura of twelve months' duration. This began on the left ankle and extended slowly during the year, appearing on both ankles and knees, the left thigh and the left forearm. He gave a history of a visual defect for the past ten months, associated with repeated retinal hemorrhages in the left eye.

In the course of the physical examination pseudoxanthoma elasticum was found, symmetrically involving both sides of the neck, the clavicular regions, the outer sides of the axillae, the antecubital region, the front and sides of the trunk from below the nipple line to the pubis, the anterior trochanteric areas, the inner sides of the midthighs, the popliteal spaces, the dorsal surface of the glans penis and the inner surface of the middle third of the lower lip. The patient was aware only of an altered condition of the skin of the trunk, which was called to his attention about five years ago as "loose skin," when it was observed in the course of an examination for life insurance.

The regions involved exhibit the disorder in various stages, from the primary lesions varying in size from that of a pinhead to that of a small split pea, which

are velvety, soft, ivory-yellow papules arranged in parallel ridges or meshlike patches, to the coarsely reticulated or meshed, relaxed and apparently atrophic terminal condition with a slate-gray color in the interstices, as shown on the trunk. Faint remains of macules of purpura are present on the left leg.

The family history is unimportant. The patient has had discharging ears since an attack of scarlet fever at the age of 5 years. The tonsils were incompletely removed by diathermy six years ago and remained infected. He has been exposed to lead for the past nineteen years.

The results of physical examination were as follows: The chest was normal. The heart was normal except for a pulmonary systolic murmur at the base. The blood pressure was 124 systolic and 84 diastolic. The liver was palpable but not enlarged; the spleen was palpable 3 cm. below the left costal border with deep inspiration and was slightly enlarged. No abdominal masses were noted. There was inguinal shotlike adenopathy. The thyroid gland was not enlarged. There was perforation of both ear drums, with discharge. The teeth had all been extracted. The reflexes were normal. The tourniquet test produced petechiae and ecchymosis within five minutes.

Examination of the eyes (performed on November 24 by Dr. Neff) showed: In the right eye, there were multiple small areas of chorioretinitis and obliteration of several blood vessels. In the left eye, the vitreous body was filled with vitreous opacities. The nerve head was normal in appearance. The macular region was obliterated by round retinal hemorrhages, in the center of which choroidal pigment showed through, the involved area being about the size of the nerve head. There were many fresh retinal hemorrhages.

The laboratory findings were as follows: The blood count was normal. The bleeding time was three minutes; the coagulation time, four minutes. The platelets numbered 290,000. The fragility test of the erythrocytes was essentially negative. There was complete clot retraction in twenty-four hours. The stippled cell count was 0.2 per cent. The Wassermann reaction of the blood was negative. The blood sugar was 95 mg. per hundred cubic centimeters, and the nonprotein nitrogen, 30 mg. Urinalysis revealed nothing abnormal except 7 coarse granular casts and 2 hyaline casts per ten low power fields.

On microscopic examination the epidermis and papillary layers showed no appreciable change. The middle cutis showed characteristic clumped, frayed and segmented groups of elastic fibers, which in many instances were reduced to small granular clumps. In the most severely affected areas the collagenous fibers stained more lightly than normal, were slightly swollen and had a ground glass appearance. Inflammatory cellular infiltration was absent, and the glandular structures were intact.

DISCUSSION

Dr. Elmore B. Tauber, Cincinnati: One thing that struck me in regard to these patients was the ocular disturbance. I previously observed two patients in whose cases a diagnosis of pseudoxanthoma elasticum had been made by the ophthalmologist before I saw them. The diagnosis was proved afterward, but the patients came under observation originally because of the ocular rather than because of the cutaneous condition.

Dr. Udo J. Wile, Ann Arbor, Mich.: I think that the conditions presented by these patients are much alike in some respects and different in one respect. Both have characteristic lesions more or less in the typical sites of pseudoxanthoma elasticum, but Dr. Foerster's patient has extensive macular atrophy all over the abdomen, and in this area I can find no residue of the pseudoxanthoma lesions. Perhaps Dr. Foerster can state, as he is familiar with the literature, whether pseudoxanthoma elasticum has been cited as one of the causes of macular atrophy. Leaving out the lesions of the thigh, where the xanthoma lesions were present, the condition could well pass as macular atrophy.

Dr. Karl G. Zwick, Cincinnati: Thanks to the courtesy of Dr. George B. Heidelman, an ophthalmologist, I recently had the opportunity of examining the

skin of a patient with Knapp's "angioid streaks" for the simultaneous occurrence of pseudoxanthoma elasticum. In contradistinction to Dr. Tauber's as well as to Lewis and Clayton's findings, I was unable to establish the presence of pseudoxanthoma elasticum in Dr. Heidelman's patient. The patient is being kept under observation for further ocular and cutaneous development.

Dr. Foerster's patient presents areas with reticulated atrophic lesions of a pale, almost white color. The patient stated that these are older lesions which were formerly distinctly yellow. This is of interest with reference to the views of some authors that involution of the condition apparently has not been observed.

Dr. Ruben Nomland, Chicago: Dr. Klein found angioid streaks in the left eye of Dr. Foerster's patient today.

Dr. O. H. Foerster, Milwaukee: Dr. Tauber spoke of such patients coming first to the ophthalmologists and of the diagnosis being made originally by them. This patient came to me because of purpura, and in the course of examination the other disorder was found. It is known that during five years there has been a change in the condition over the front of the trunk, and, as Dr. Zwick states, this apparently represents a terminal change. I think that one case has been reported in the literature which showed meshlike atrophy as a terminal change. That is the only other one of which I know. Examination of the eyegrounds in my case showed indefinite angioid streaks, so far as the ophthalmologist could determine, and I was much interested to have Dr. Klien demonstrate the angioid streaks today.

Kaposi's Hemorrhagic Sarcoma. Presented by Dr. J. H. Mitchell, Chicago, and Dr. H. E. Michelson, Minneapolis.

This woman, aged 61, states that a solitary lesion first appeared in the first interspace of the right hand. This was removed and sectioned by Dr. Michelson one year ago, and she was given radiotherapy at that time. There has been no recurrence on the right hand. About three months ago new lesions appeared in the left palm. A plantar wart was removed (J. H. M.) from the left sole in October 1930. The soles are free from lesions at present.

The patient has had several abdominal operations, but her general health has remained good.

On examination of the blood, the hemoglobin was 85 per cent. The leukocytes numbered 8,200. The differential count showed: polymorphonuclear neutrophils, 51 per cent; lymphocytes, 47 per cent; basophils, 2 per cent, and transitionals, 2 per cent. The blood pressure was 162 systolic and 90 diastolic.

The urine contained no albumin or sugar and gave an acid reaction.

DISCUSSION

Dr. C. W. Finnerud, Chicago: I was much surprised when I saw the histologic picture of Kaposi's sarcoma, changes which I did not expect to find from what I saw grossly.

Dr. Jeffrey C. Michael, Houston, Texas: I think that the patient has sarcoid; the histologic picture looked more like that than anything else. I saw a dense infiltrate and a few giant cells which I associate with sarcoid and not with Kaposi's sarcoma.

Dr. J. H. Mitchell, Chicago: By way of explanation, I wish to say that this history with the diagnosis was prepared and typed before the section arrived. As I was leaving the office the section arrived by mail. I thought of Kaposi's disease when I first saw the patient about ten days ago, and my impression was confirmed by the report that Dr. Michelson made of his examination of the tumor.

Several comments were made on the small keratotic lesion on top of the largest one. When I saw that lesion first it was smooth and shiny; for the sake

of learning what might be in it I punctured it with a needle, and this is the result of the puncture. The discussion of the histologic features is to be left entirely to Dr. Michelson, for he made the section.

DR. H. E. MICHELSON, Minneapolis: When a patient does not present more clinical evidence than this woman does it is hazardous to make a diagnosis. Professor Joseph told Dr. Freeman years ago that without lesions on the feet a diagnosis of Kaposi's disease was questionable. A biopsy, however, I believe, confirms the clinical diagnosis that we ventured. The patient responded well to arsenic and roentgen therapy. I think that with these findings we have all the elements necessary to substantiate the diagnosis. Dr. Michael mentioned sarcoid. I have never seen or heard of sarcoid in the palm, but I presume there is no reason why it cannot occur there.

KERATOTIC ANGIOMA. Presented by DR. S. W. BECKER, Chicago.

This girl, aged 3 years, presents a lesion on the lower part of the right leg which has been present since birth. There has been no marked increase in size; the lesion has bled occasionally when traumatized. On the outer aspect of the lower part of the right leg are an elevated, dusky-red, markedly keratotic plaque 3 by 5 cm. and a similar lesion 1 by 1 cm. adjacent to it.

Carbon dioxide snow, radium therapy and electric cauterization had been used, with only slight improvement.

A section is presented.

HEMANGIOMA CAVERNOSUM OF THE EAR; ANGIOFIBROSARCOMA (?) OF THE NECK. Presented by DR. E. P. ZEISLER, Chicago.

This man, aged 29, presents an angioma of one side of the face, involving the ear and the cervical region, which has been present since birth. Fifteen years ago the angioma of the ear was treated by injections of boiling water (eighteen) and tying off of a blood vessel in the neck. This was followed by moderate improvement. During the last five years the tumor of the neck has gradually enlarged to its present size.

There is a huge, lobulated deformity of the ear, with fibrous tissue replacing cavernous tissue. On the left side of the neck is a freely movable, deep red, irregularly lobulated mass the size of an orange, with a peculiarly mamillated appearance, suggesting a cavernous hemangioma with fibrosis and possibly sarcomatous change.

The patient was seen for the first time yesterday, and no biopsy material had been secured. He is presented for suggestions as to whether radium therapy or surgical intervention is indicated.

DISCUSSION

DR. RICHARD L. SUTTON, Kansas City, Mo.: The first patient, the child, has a keratotic angioma. One might discuss the classification of angiokeratoma, but I should not put this condition in that group. I think it is only an angioma. I did not see the second patient.

DR. S. W. BECKER, Chicago: The condition which this girl presents is somewhat different from the ordinary angioma in that it is more of an angiomatosis. There is a profuse growth of blood vessels throughout the tissues, about the sweat glands and even in the adipose tissue. The picture is similar to that presented by a patient shown by Dr. Cornbleet a month or so ago, in which the tissues showed this angiomatous change in addition to deep lipomas. Such conditions are difficult to treat. I do not know how deeply the angiomatous change goes, but as there is considerable vascular development, it is obvious that deep treatment of a destructive type must be used.

DR. E. P. ZEISLER, Chicago: I showed my patient merely to bring out the different types of clinical picture often seen in association with angioma, particularly the cavernous type. What the exact change is that has taken place in the tumor of the cervical region cannot, I think, be reported without a biopsy, and that will be done later. Various possibilities present themselves: fibrosarcoma, angiosarcoma and angiosarcoma protuberans. I shall report on the case at a later date.

NOTE: Biopsy on tissue from the neck was reported as showing "lymphangioendothelioma."

PITYRIASIS RUBRA PILARIS. Presented by DR. J. H. MITCHELL and DR. RUBEN NOMLAND, Chicago.

This girl, aged 10 years, presents a disorder of five weeks' duration. She had had no definite illness but had felt below par for several months. There has been no cough and no evident loss of weight. The lesions appeared almost simultaneously on the palms and soles, and later on the elbows and knees. Recently the face and chest have become involved.

When first seen, on Jan. 10, 1934, there were mortar-like deposits on the brows and chin and especially about the ears. The chest was covered with follicular keratotic cones, but none were seen on the proximal phalanges of the fingers. The palms and soles and, to a lesser degree, the elbows and knees were symmetrically much thickened. The child appeared pale and was so nervous that photography and laboratory examinations were difficult. The general physical examination gave negative results except for pustular vaginitis. There were many gram-positive cocci, some of which were in short chains. Brain broth culture of the vaginal discharge contained long chains of cocci and staphylococci.

The patient was given one-half skin unit of roentgen rays to the palms and soles, salicylated oil and viosterol, followed by marked improvement in the general condition. The face is now practically clear.

On January 10, the hemoglobin was 82 per cent. The leukocytes numbered 12,000 per cubic millimeter. The differential count showed: Polymorphonuclear neutrophils, 47 per cent; small lymphocytes, 20 per cent; large lymphocytes, 23 per cent; eosinophils, 1 per cent; basophils, 1 per cent, and transitionals, 8 per cent. The blood pressure was 104 systolic and 46 diastolic. On January 13, the urine showed a trace of albumin and no sugar. The reaction was acid. There were many pus cells and calcium oxalate crystals. The leukocyte count was 11,800. On January 18, the leukocyte count was 10,200.

DISCUSSION

DR. EVERETT S. LAIN, Oklahoma City: I agree with the diagnosis. The condition seems to be characteristic, and it appears that such cases are relatively more frequent in my section of the country. Two or three cases have been shown at meetings in Oklahoma. The interesting feature in this case is that the condition apparently is undergoing changes. Moreover, the follicular horny plugs are not present over the dorsa of the phalanges. That has been true in many other cases I have seen. They may be present in certain stages of the disease but not constantly. Four or five years ago I attended a meeting in Chicago at which some one stated that a French dermatologist had attempted to treat patients with this condition by means of filtered roentgenotherapy over the thymus gland. At that time I was treating a patient with a striking case. After returning home I gave my patient two or three roentgen ray treatments at intervals of four weeks. He showed marked improvement. He still has the disease, but it has been much modified, and he insists that the roentgenotherapy deserves the credit.

DR. O. H. FOERSTER, Milwaukee: It always seemed to me that in the diagnosis and recognition of these cases in the early stages the mortar-like scale that is seen on the scalp is of some diagnostic assistance. The history usually shows

that for a variable length of time preceding any cutaneous lesions there is the peculiarly thick, mortar-like scaling of the scalp that is present in this case. The involvement of the palms and soles, the bandlike lesions and the patches on the forearms are suggestive of the disease. The flatness of the nails, even though they show no pitting, and the patch on the left anterior axillary fold, I think, clinch the diagnosis.

DR. WILLIAM ALLEN PUSEY, Chicago: I had no semblance of a clear idea of the case until Dr. Foerster described it, but I cannot sit still and hear a diagnosis of Devergie's disease made. If I remember, the outstanding characteristic is fairly well marked keratoderma of the palms and soles. In addition, there are a defect in the development of the nails and one or two patches on the knees and elsewhere that suggest a congenital defect. There are lesions that may be those of epidermolysis bullosa, and there apparently is a congenital defect in the nails. I am familiar with the fact that it is said that the condition has been present only for a few months, but I should not rely on the statement of the patient that it has lasted no longer than that, and I suggest that before making a diagnosis of Devergie's disease one consider hereditary keratoderma. I think this a more likely diagnosis than pityriasis rubra pilaris.

DR. C. W. FINNERUD, Chicago: It may be that I examined the patient too hurriedly, but the diffuse keratoderma of the palms and soles, the seborrhea of the face and scalp and the psoriasiform lesions on the knees and elsewhere caused me to consider the condition characteristic Devergie's disease in spite of the lack of follicular papules on the backs of the hands. In three severe cases of this disorder, two of which were shown before the Chicago Dermatological Society, the condition cleared up in less than a year under treatment with thyroid taken to the tolerance of the patient. The patients presented were young women. The patient in a third case was not shown but Dr. Ebert saw her with me when she was in an extremely serious condition and again after the lesions had practically cleared; in this case thyroid alone apparently was responsible for the "cure." In all of these cases there was a very low basal metabolic reading, something like —28 or —30 in the beginning.

DR. RICHARD L. SUTTON, Kansas City, Mo.: I agree with Dr. Pusey. I recall the case of an Italian boy who came to me seven or eight years ago with a similar history. When I last saw him, several years later, Devergie's disease had not developed.

DR. JOHN BUTLER, Minneapolis: The condition on the hands suggests psoriasis. I do not think that it is pityriasis rubra pilaris. The follicular plugging on the backs of the fingers may or may not be present in pityriasis rubra pilaris.

DR. E. P. ZEISLER, Chicago: The patient presents involvement of the palms and soles and psoriasiform plaques, and I think that true Devergie's disease will develop within the next few months.

DR. LOUIS A. BRUNSTING, Rochester, Minn.: I have recently observed three cases of Devergie's disease in which the condition developed during adult life. In one there was a resemblance to psoriasis except for the histopathologic picture. In all three there was considerable improvement from sweating induced by pilocarpine.

DR. KARL G. ZWICK, Cincinnati: The absence of lesions on the backs of the hands does not prompt me to reject the diagnosis of pityriasis rubra pilaris (Devergie), which in this country is still looked on as being, without exception, identical with Kaposi's lichen ruber cuminatus. But I have asked myself whether the condition on the palms and soles should not lead one to consider the diagnosis of keratoderma palmare et plantare.

DR. J. H. MITCHELL, Chicago: I have no doubt as to the diagnosis. I suggest that Dr. Pusey read the description of this disease in his textbook. I have read the reports of a number of cases recently, and this is a commonplace syndrome

in a child of this age. I even hesitated to show it but was urged by Dr. Ebert to do so. I presented the child with this diagnosis without any hesitation. Dr. Foerster seems to agree with me in regard to the mortar-like deposits about the ears and on the scalp, which were much more pronounced when I first saw the child. The use of salicylic ointment has removed a good deal of these deposits. The presence of the cones on the legs is definite. The child is too young and the duration too short for the follicular lesions to have developed on the hands. The interesting thing to me is that she has acute vaginitis and that streptococci were found in the vaginal discharge. Whether this has anything to do with the cutaneous condition I do not know. The leukocytes have numbered about 12,000 on repeated counts. Tests for tuberculosis were negative.

All of the members are familiar with the difference of opinion between Pautrier and Gougerot regarding the etiology of pityriasis rubra pilaris. Gougerot and Milian are convinced that the disease is of tuberculous origin, but Pautrier is not convinced of this. Gougerot claims to have been the only one who has succeeded in curing this disorder. He now has cured it in cases resulting from the use of Vaudremer's tuberculous vaccine. I shall keep the child under observation as closely as I can, and if the condition proves to be something else I shall eat crow if necessary with Dr. Pusey.

AMYLOIDOSIS (LOCALIZED, LICHEN) OF THE SKIN. Presented by DR. RUBEN NOMLAND and DR. F. V. PLUMMER, Chicago.

This patient was presented at the October 1933 meeting of the Chicago Dermatological Society, and the history was published in the transactions of that society (ARCH. DERMAT. & SYPH. **29**:470 [March] 1934).

DISCUSSION

DR. H. E. MICHELSON, Minneapolis: The members are fortunate to see this excellent example of localized amyloidosis. We have seen several conditions due to metabolic disturbances this afternoon, and this is one of them. In localized amyloidosis there is probably no systemic disturbance, although the congo red test showed considerable absorption.

DR. RICHARD L. SUTTON, Kansas City, Mo.: I wish to congratulate the presenters. In many of these cases one is likely to diagnose the condition as hypertrophic lichen planus.

DR. WILLIAM ALLEN PUSEY, Chicago: I wish to add my praise of the younger generation. One who has reached Dr. Sutton's age and mine would probably pass such a case as one of hypertrophic lichen planus, but the presenters have recognized the condition as something else.

DR. OLIVER S. ORMSBY, Chicago: In Dr. Nomland's investigation of this case he did some original work in staining, and I should like to have him report on that work.

DR. RUBEN NOMLAND, Chicago: I wish to correct a misapprehension of Dr. Michelson's as to the intravenous congo red test. There was only a 15 per cent loss of dye from the serum in one hour, which is within normal limits. The patient has no generalized amyloidosis, as evidenced by that test and by complete examination. The interesting thing was the local vital staining with congo red solution. In September, 1.5 cc. of 1.5 per cent congo red was injected under the skin near the amyloid nodules, and one can still see the red-stained nodules that took up the congo red. The nodules did not take it up after intravenous injections. In addition to the Bennhold test, the patient was given congo red intravenously, 15 cc., several times, and none was demonstrated as being taken up by the amyloid nodules from the blood stream. I did not have sufficient courage to inject congo red into the femoral artery, but in my next case I think that I am going to inject it and see if it will be taken up through the blood stream.

IDIOPATHIC ATROPHY WITH POIKILODERMA-LIKE CHANGES? MYCOSIS FUNGOIDES? Presented by DR. E. A. OLIVER, Chicago.

This patient was presented at the February 1933 meeting of the Chicago Dermatological Society, and the history was published in the transactions of that society (ARCH. DERMAT. & SYPH. **28**:438 [Sept.] 1933).

The patient feels better this year than he did in 1933.

On Jan. 20, 1934, the erythrocyte count was 5,300,000; the leukocyte count, 9,100, and the hemoglobin, 92 per cent. The differential count showed: small mononuclears, 16 per cent; monocytes, 15 per cent; polymorphonuclears, 68 per cent, and eosinophils, 1 per cent. The erythrocytes were of good color and of normal size and shape; there was no polychromatophilia.

DISCUSSION

DR. UDO J. WILE, Ann Arbor, Mich.: I offer a diagnosis of mycosis fungoides in spite of the fact that the lesion on the arm shows some atrophy. There is considerable infiltration in the lesions on the back and also on the right flank, and the section shows changes which are compatible with granuloma fungoides. The only unusual feature is that the patient says that he has had little or no pruritus. I do not think the condition can be poikiloderma, for those lesions are usually uniform in distribution and not patchy as these are. I think that fungoid tumors will develop later.

DR. C. W. FINNERUD, Chicago: I saw the sections when the patient was presented before, and if I am not mistaken the diagnosis of mycosis fungoides was considered because of the histologic changes in one of the papillomatous lesions on the trunk.

DR. RICHARD S. WEISS, St. Louis: I do not think that the absence of pruritus is absolutely against the diagnosis of mycosis fungoides. When I examined the patient, I gained the impression that there was no pruritus, and he said that there was practically none. I have observed several cases in which pruritus was not marked and later mycosis fungoides became evident. I agree with Dr. Wile in the diagnosis.

DR. KARL G. ZWICK, Cincinnati: As the diagnosis of mycosis fungoides has been considered, I draw attention to the patient's statement that he does not suffer from pruritus and that his condition improves when he perspires freely during the summer.

Although spontaneous remissions, progressing almost to cure, have been described in mycosis fungoides, I do not recall that the recurrence of remissions at regular intervals has been noted.

It would interest me to learn if any changes in the sweat glands are observed on histologic examination.

DR. RICHARD S. WEISS, St. Louis: I do not think that the point which Dr. Zwick brought out is apropos, for I have seen mycosis fungoides in the premycotic stage involute again and again, and the statement of the patient that the condition is better at times should not invalidate that diagnosis.

DR. OLIVER S. ORMSBY, Chicago: Last June at the clinical meeting of the American Dermatological Association I presented a woman who had, scattered over the body, patches much like these which were palm-sized and larger, atrophic and scale-covered and resembled patches of idiopathic atrophy of the skin. In addition, she had several plaques which were typical of mycosis fungoides and which presented the histologic picture of that disease. So it is possible to have lesions of this type in mycosis fungoides. That patient died of pneumonia a few days ago. Such cases show the remarkable versatility of the disease classed as mycosis fungoides.

DR. E. A. OLIVER, Chicago: I admit that Dr. Wile's suggested diagnosis of mycosis fungoides is one of the best that has so far been given, but I am not yet convinced that it is the correct one.

I have seen this patient at intervals during the last year. He is not a great deal worse than he was a year ago. There are a few new smaller atrophic areas, but the large lesions have changed little. His general health is better than it was. He has never complained of pruritus. These facts refute the generally accepted ideas of mycosis fungoides.

A Case for Diagnosis (Universal Erythroderma with Lichenification?). Presented by Dr. E. P. Zeisler, Chicago.

This boy, aged 16, states that in January 1930 his face became red; this was followed by desquamation, and the condition then spread over the entire body and was followed by pigmentation. He was hospitalized (Michael Reese Hospital) in August 1931. At that time there were universal erythroderma, lichenification, almost black pigmentation of the hands, feet, axillae and flexures and acanthotic areas in the axillae, palms and soles which suggested acanthosis nigricans. Moderate lymphadenosis and an enlarged spleen were noted.

The Wassermann reaction of the blood was negative. The erythrocyte count was 4,600,000; the leukocyte count, 12,000, and the hemoglobin, 80 per cent. The differential count showed: polymorphonuclear neutrophils, 57 per cent; eosinophils, 16 per cent; basophils, 1 per cent; lymphocytes, 13 per cent, and monocytes, 13 per cent. The blood sugar was 69 mg. per hundred cubic centimeters; the nonprotein nitrogen, 25 mg., and the cholesterol, 212 mg. The sugar tolerance curve was normal.

Roentgenographic examination of the gastro-intestinal tract gave negative results, revealing no neoplasm; the sella turcica and the thymus were normal.

The basal metabolism was + 6. The test of kidney function (phenolsulphonphthalein) showed excretion as follows: first hour, 40 per cent; second hour, 10 per cent; third hour, a trace. The urine was reported to contain arsenic (amount not determined).

Marked improvement followed the use of sodium thiosulphate orally, baths and ultraviolet therapy. Since then there have been acute exacerbations at varying periods, requiring hospitalization. The most marked relief was obtained from compound phenyl salicylate ointment.

Examination of the blood in 1932 showed: leukocytes, 15,400; polymorphonuclear neutrophils, 61 per cent; eosinophils, 16 per cent; lymphocytes, 9 per cent, and monocytes, 14 per cent.

The patient is presented with a provisional diagnosis of universal erythroderma (arsenical? lymphoblastomatous?) with lichenification, possibly combined with acanthosis nigricans of the juvenile type.

A biopsy specimen is presented.

DISCUSSION

Dr. E. P. Zeisler, Chicago: I saw this patient first in 1931 and hospitalized him. He then had generalized lichenification of the entire body, with almost black pigmentation which was most marked around the axillae and groins, and keratosis of the palms and soles. My associates and I made many studies but were unable to arrive at any conclusion as to the character of the erythroderma. We considered a juvenile type of acanthosis nigricans. He had some attacks of abdominal pain which suggested an abdominal neoplasm, but we have never been able to find any evidence of such a growth. He has had repeated roentgenographic examinations. He has gained in weight. Under treatment with sodium thiosulphate many of the keratotic lesions regressed, those on the palms and soles disappeared and the general cutaneous condition showed marked improvement. He has had on several occasions in the past three years attacks of acute erythematous lesions on the face, scalp and body. Within the last six months the peculiar lichenification of the legs, which looks like lichen obtusus corneus, appeared. The histologic section does not show amyloidosis; there is some pigment as well as chromatophores with some acanthosis. The patient was first

seen in the clinic of the Rush Medical College, and the physicians there may be able to throw some light on the early findings. If I had to make a diagnosis I should say that the condition may be juvenile acanthosis nigricans.

DR. RUBEN NOMLAND, Chicago: The patient was seen at Rush Medical College when the disorder was in an early stage. At that time many of the lesions were discrete lichenoid papules, and the diagnosis of lichen planus was thought the most likely until biopsies were made. Later the more or less general dermatitis appeared; Dr. Mitchell then said that the condition probably was malignant erythroderma, and I agreed with him. I still hold that opinion.

DR. RICHARD L. SUTTON, Kansas City, Mo.: Have any cutaneous sensitivity tests been made? The case gives me the impression of a sensitization problem, and that type of investigation might uncover the underlying disorder.

DR. LOUIS A. BRUNSTING, Rochester, Minn.: I was about to suggest for several reasons that the condition be considered from the standpoint of a sensitization dermatosis, particularly of the group poorly called "generalized neurodermatitis." There are indications of atopy. First, the boy is of adolescent age, and the symptoms have been present since the age of 12, beginning with involvement of the flexor surfaces of the elbows and knees and the face and neck and proceeding through remissions and relapses to the present universal distribution. The chief objective signs now are lichenification and pigmentation, although there are many groups of follicular papules. The use of arsenic or roentgen rays might be considered as a contributing factor in the development of this striking degree of pigmentation. The boy has a history of vasomotor rhinitis, and his mother has hay fever. Sensitization in the form of contact dermatitis is not likely except as a possible added factor to the inherent atopic sensitivity. The microscopic picture reveals that the epidermis is chiefly involved, with marked acanthosis and spongiosis in the upper cutis. There is considerable edema, such as one would find in urticaria, and the infiltrate is nondescript, certainly not suggestive of lymphoblastoma. Protein tests by the direct method are out of the question because of the universal involvement. However, a test by the method of passive transfer of Prausnitz and Küstner should be made in an attempt to demonstrate circulating reagins. We have under our care at present a woman about 29 with exactly the same problem. Lymphoblastoma and exfoliative psoriasis were considered at first but were ruled out on the basis of the histologic observations. By passive transfer the patient was found sensitive to a number of inhalants, as well as to wheat and milk, and by carefully controlling her environment and using an elimination diet there has been a gradual steady improvement in her condition.

I think that dermatologists are inclined to lean backward a little in questions of allergy, partly because they have not been able to reconcile the various laboratory tests which have been reported with the clinical condition of the patient and partly because of the overenthusiastic reports of certain allergists who are not well grounded in dermatology. Laboratory tests should be made under the supervision of the examining physician and should be interpreted to meet the demands of each particular case.

DR. S. W. BECKER, Chicago: I think that the hyperpigmentation is due to the boy's capability of retaining an enormous amount of pigment in the chromatophores and that it is entirely secondary. I did not see any evidence of acanthosis nigricans, for lesions in the axillae are not present. There are some vegetating lesions, but I think that the infiltrate is merely lymphocytes. I believe the condition is lymphoblastoma and not neurodermatitis.

ECTODERMAL DEFECT. Presented by DR. E. A. OLIVER, Chicago.

This youth, aged 18½, states that he was normal until the age of 6 years and presents a photograph taken at that time. He then had a fall which fractured his skull and evidently caused hydrocephalus to develop. From that time normal development ceased. He is a senior in high school and will graduate in June. His height is 4 feet and 4 inches (132.08 cm.), and his weight, 70 pounds (31.8 Kg.).

He spent two days in the hospital in August 1930. The laboratory findings at that time were as follows: The Wassermann and Kahn reactions were negative. The erythrocyte count was 3,910,000; the leukocyte count, 6,300, and hemoglobin, 65 per cent. The urine was normal. The basal metabolic rate was + 34.

He states that perspiration is normal and that he perspires freely when warm. There is no history of any one in his paternal or maternal family having been similarly affected.

The patient has a large hydrocephalic type of head. There is a marked paucity of scalp hair; the eyes are set close together, and there is wrinkling about the eyes, the nose and the angles of the mouth. The hands are spadelike, and there is a deformity of all the nails of the fingers and toes. There are three malformed teeth in the upper and three in the lower jaw. The jaws are very narrow and are slightly deformed.

The skin is not dry but smooth and soft. The mentality is above par; in fact, he seems unusually bright.

DISCUSSION

DR. PAUL A. O'LEARY, Rochester, Minn.: I believe that the condition belongs in the group of chondrodysplasias rather than in the group of congenital ectodermal defects. The localized sweating and the large central incisors instead of small, pegged-point teeth are against the latter diagnosis. In addition, the oxycephaly that the boy presents is not, as a rule, seen in persons with a congenital ectodermal defect, in whom a large, prominent forehead is more commonly noted.

DR. E. A. OLIVER, Chicago: Although I dislike to disagree with Dr. O'Leary, I cannot accept his diagnosis. There is pronounced dental aplasia. This boy has only three teeth in the upper and three in the lower jaw. The hair on his scalp is scanty, to say the least; he has a deformity of all the finger-nails and toe-nails. There is atrophy of the skin of the face, producing wrinkles about the mouth, nose and eyes. The picture is similar in many respects to that in the case reported by McKee and Andrews (ARCH. DERMAT. & SYPH. **10**:673 [Dec.] 1924) except that the patient perspires freely.

The interesting feature is the fact that up to the age of 6 years, according to his story and as revealed by his photographs, he was apparently normal. At the age of 6 he fell and injured his head rather badly. He does not know whether or not his skull was fractured. From that time he began to acquire these characteristics.

No one in his mother's or father's family has ever shown similar symptoms.

LUPUS MILIARIS DISSEMINATUS FACIEI. Presented by DR. OLIVER S. ORMSBY, Chicago.

This patient was presented at the December 1933 meeting of the Chicago Dermatological Society, and the history was published in the transactions of that society (ARCH. DERMAT. & SYPH. **30**:137 [July] 1934).

DISCUSSION

DR. H. E. MICHELSON, Minneapolis: I believe that this patient has classic lupus miliaris disseminatus faciei. The work of Arndt should be referred to in association with this disease, for he has definitely shown that the lesions are not, in their entirety, follicular. The clinical course and the histologic observations bring the disease closer to lupus vulgaris than to tuberculid.

DR. FRED R. SCHMIDT, Chicago: The members are greatly indebted to Dr. Ormsby for showing this classic case of cutaneous tuberculosis. I saw Kyrle in one case give a 1:1,000,000 dilution of old tuberculin; the patient went into profound shock, and the focal and systemic reactions were severe. He advised starting with very small doses. I have seen patients improve greatly under treatment with old tuberculin.

KERATODERMA BLENNORRHAGICUM. Presented by DR. THEODORE CORNBLEET and DR. E. R. PACE, Chicago.

This white man, aged 65, presents an eruption of the soles which, he states, has developed during the past two months; coinciding with this was a second attack of arthritis, with active gonorrheal urethritis. He stated that his initial gonorrheal infection occurred fifty years ago but that there was a reinfection five or six years ago, about two months after which there developed arthritis of the right hip, right wrist and left knee, which subsided in two months. The present recurrence of the arthritis involved the right hip, left wrist, right ankle, left knee and shoulder, and at the same time the eruption on the soles was first noted.

There are now discrete, horny excrescences and diffuse keratotic patches on both soles, including the toes, more extensive on the right foot. The larger lesions tend to be conical. The individual lesions have no inflammatory areolae. Shallow, clean, pink erosions remain following removal of the superjacent keratotic material. The nail beds are covered with a granular keratotic material, which raises the nail plate from the bed; the nails themselves are thickened and opaque. The palms are free from lesions.

The urethral discharge showed gram-negative intracellular diplococci. The leukocyte count was 16,100, and the differential count showed: polymorphonuclear neutrophils, 60 per cent; lymphocytes, 24 per cent; monocytes, 15 per cent, and eosinophils, 1 per cent. The sedimentation was 18 mm. in ten minutes. The Kahn test was negative.

The temperature has varied from 98 to 102 F.

Biopsy on two specimens taken from a typical conical lesion revealed in each a separated horny piece composed of parallel layers of parakeratotic scale and thickened base. The epidermis was acanthotic and showed a thick granular layer, moderate edema and an intact basal layer. The corium was edematous and vascular, showing a diffuse infiltrate of lymphocytes and histiocytes and fewer plasma cells. In one place there was an edematous polyp-like projection covered by thin epidermis.

PARAPSORIASIS LICHENOIDES. Presented by DR. OLIVER S. ORMSBY and DR. M. H. EBERT, Chicago.

This steel worker, aged 47, presents a cutaneous disorder which is most marked on the covered portions of the body. He states that the skin has always been dry. In July 1931 he tended a furnace, working in intense heat. During this period he perspired normally, but his skin became dry and scaly. He thinks that there has been slight improvement in the condition since that time. He has never worked with chemicals or handled tar and denies having taken Fowler's solution. He has received local treatment but no violet ray or roentgen ray therapy. There are no subjective symptoms, and his general health appears to be good.

There is dry pityriasic scaling over the covered portions of the body. In this scaly surface there is a background of flat-topped maculopapules or papules the size of a small pinhead, with a poor outline, very slightly elevated and in some areas having a reticulate arrangement. Some are grayish-brown and some pinkish; definite telangiectasia can be seen in some of them. The trunk is covered with papules varying in size from that of a pinhead to that of a match head which are slightly elevated and resemble seborrheic warts but are more keratotic. These lesions are most numerous and largest on the chest. In some areas the skin when relaxed seems to fall into atrophic wrinkles. The palms are dry, with slight keratotic scaling.

A biopsy specimen was taken from a match head-sized papule which resembled a seborrheic wart in the middle of the back and from a brownish, keratotic papule the size of a large pinhead over the left scapula. The first section showed marked hyperkeratosis with pronounced pluggings of the openings of the hair follicles and sweat ducts. The epidermis was thin over the tops of the papillae, and the rete pegs were somewhat elongated. The stratum granulosum was poorly devel-

oped or absent. There was considerable edema of the rete mucosa in small areas. The papillae were edematous and in some places contained dilated capillaries filled with red cells. There was a mild inflammatory infiltration about the vessels of the reticular and papillary layers and about the hair follicles, made up chiefly of lymphocytes. The second specimen showed similar changes, except that the hyperkeratosis was not so distinct and nucleated areas alternated with nonnucleated —i. e., patchy parakeratosis. In some places the edema of the rete was marked.

DISCUSSION

DR. UDO J. WILE, Ann Arbor, Mich.: I think that the condition is general parapsoriasis and that it would be better to call it that than pityriasis lichenoides chronica. The cases of the latter disease with which I have been familiar have not been so profuse. Furthermore, the lesions are usually of a brighter, redder color than these.

Pityriasis lichenoides chronica presents so sharp and distinct a picture that I think it can be differentiated clinically from other forms of parapsoriasis.

DR. JEFFREY C. MICHAEL, Houston, Texas: I should like to know whether the patient has been treated because of Ehrmann's statement that such eruptions respond to ultraviolet radiation and pilocarpine.

DR. OLIVER S. ORMSBY, Chicago: The patient has just come under our observation. The records show that he has had no roentgenotherapy or phototherapy and no internal treatment. However, in a number of other cases we have had better clinical results with phototherapy than with other methods.

DR. FRED R. SCHMIDT, Chicago: No dermatologist thinks of seeing vesicles grossly in this condition, but under the microscope one sometimes observes marked dyskeratosis associated with vesicles. The vesicles may never come to clinical expression, and when they are noted under the microscope they are situated intradermally.

NEVI, MULTIPLE. Presented by DR. M. H. EBERT and DR. M. J. WOLFF, Chicago.

This patient was presented at the November 1933 meeting of the Chicago Dermatological Society, and the history was published in the transactions of that society (ARCH. DERMAT. & SYPH. **29**:765 [May] 1934).

DISCUSSION

DR. PAUL A. O'LEARY, Rochester, Minn.: Clinically the condition resembles that in a case of multiple ganglioneuroma which I presented before this organization in October 1932 and at the clinical meeting of the American Dermatological Association on June 10, 1933, and which has been reported in full in the ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY (**29**:26 [Jan.] 1934). There are, however, several distinct differences, sufficient to lead me to believe that this is not ganglioneuroma. The patient has many verrucous lesions scattered among the papules, while our patient did not show any warty nodules. Moreover, Dr. Ebert's case does not reveal the fawn-colored papules, elicited by pressure, which are a prominent feature of ganglioneuroma. Dr. Ebert's inability to demonstrate the presence of ganglion cells with a silver stain is the outstanding point against the diagnosis of ganglioneuroma. At the present time that diagnosis is impossible on the basis of clinical appearance alone and can be made only by the recognition of ganglion cells.

DR. S. W. BECKER, Chicago: Through Dr. Ebert's kindness my associates and I had an opportunity to study this case. We performed the dopa test on some of the sections, and all of the nevus cells, even the deepest, showed a positive reaction. They also contained a small amount of melanin. I think that there

is no doubt that this case is a rather unusual one of acquired pigmented nevi. We have not completed our study but hope to throw some light on the origin of the tumors.

DR. M. H. EBERT, Chicago: In view of the clinical resemblance of this condition when it was first seen to that in the case of neuroganglioma presented by Dr. O'Leary, it is interesting to note that Masson and Pautrier believe that the cells in this type of nevus are derived from the *cellules claires* of the epidermis, which are analogous to the touch cells of Merkel and hence are part of the peripheral nervous system. We are making further detailed histologic studies of the material.

A CASE FOR DIAGNOSIS. Presented by DR. F. E. SENEAR, Chicago.

This boy, aged 4 years, presents a dermatitis which, the mother states, began as a lesion on the buttock at the age of 6 weeks. The primary lesion rapidly enlarged to cover part of the buttock, and soon afterward the eruption appeared on the feet. The child was taken to the Cook County Hospital, where he was treated with ointments, but the mother was not told what the diagnosis was. At the age of 2 years the lesions spread to the face and hands, and the child was then in the Children's Memorial Hospital for a year, without any improvement.

For the past nine months he has had photophobia. He shows symblepharon, cicatricial entropion and corneal opacities in both eyes.

The records at the Children's Memorial Hospital state that in August 1931 a diagnosis was made of dermatitis venenata; in November 1931, of epidermophytosis, and in 1931 and in February 1932, of lupus erythematosus. The child's father has had lupus vulgaris since the age of 8 years.

Examination of the blood showed: erythrocytes, 3,670,000; leukocytes, 10,550; polymorphonuclear neutrophils, 66 per cent; lymphocytes, 33 per cent; eosinophils, 1 per cent; dextrose, 100 mg. per hundred cubic centimeters; nonprotein nitrogen, 92 mg.; cholesterol, 217 mg.; carbon dioxide-combining power, 54 cc. per hundred cubic centimeters of plasma. The Mantoux test was negative.

Histologic examination of a biopsy specimen taken from the knee showed a thick, non-nucleated scale which dipped into a widened follicle. The epidermis was flattened and had lost the rete pegs. The granular layer was thin but continuous. There was intracellular edema throughout the epidermis, and the basal layer was a continuous layer of ballooned cells. The upper part of the corium was very edematous and contained many dilated blood vessels, about some of which lay many lymphocytes and histiocytes. Some of the deeper vessels had similar mantles of infiltration. The collagen fibers were thin in the upper part of the corium, and elastic fibers were absent, while lower in the corium the elastic fibers were fragmented.

DISCUSSION

DR. F. E. SENEAR, Chicago: This boy has atrophic lesions over the nape of the neck, the buttocks and the extensor surfaces of the arms, and in some places over points of pressure. The history was rather difficult to obtain, but the mother stated that the lesions started early in life and she feels that they were blisters. However, while the patient was in the hospital two or three impetiginous lesions of the face developed which she also called "blisters"; so that cast some doubt on the reliability of her earlier observation. He has shown while in the hospital no lesions which could be considered distinctly bullous, and he has lesions in regions not subjected to injury. He was in the Children's Memorial Hospital and previously in the Cook County Hospital, where, his mother said, he received no treatment and she did not know of any diagnosis having been made. He was in the Children's Hospital for a year, where first a diagnosis of eczema and later one of lupus erythematosus were considered. He was recently entered in the children's service with which I am associated and presented the picture seen at present. He has marked photophobia. Perhaps Dr. Caro can add something to the description of the histologic features.

DR. M. R. CARO, Chicago: There was atrophy of the epidermis with loss of the rete pegs. Considerable intracellular edema with ballooning degeneration of the basal layer was noted. The upper part of the corium was edematous and very vascular, and there was a slight infiltration of lymphocytes about the vessels. The infiltration was not as great, however, as is ordinarily seen in lupus erythematosus or in acrodermatitis chronica atrophicans.

DR. JEFFREY C. MICHAEL, Houston, Texas: The condition has features which suggest the diagnosis of anetodermia (Jadassohn), which is a form of primary atrophy. The individual patches began with moderate erythema and very slight infiltration. As they faded, there was left a superficial crinkled atrophy. At no stage has there been any firm infiltration such as appears in morphea, though sometimes this disease may run its course without the characteristic lardaceous stage. The child has pronounced photophobia. I do not know what significance to attach to it.

DR. ELMORE B. TAUBER, Cincinnati: I have a rather different idea. The lesions seemingly affect only the extensor surfaces; they are symmetrical, and there is atrophy. I am inclined to consider the condition as a vitamin deficiency disease. The photophobia would fit in with that conception.

DR. F. E. SENEAR, Chicago: In response to what Dr. Tauber said, the department of pediatrics reports that the child has definite evidence of avitaminosis, particularly deficiency of vitamin A, and that the photophobia is a manifestation of that disorder. I am grateful to Dr. Michael for his suggestion.

FIBROSIS (PAPULAR) OF THE PALATE; ELEPHANTIASIS OF THE UPPER LIP.

Presented by DR. A. W. STILLIANS, Chicago.

This Swedish woman, aged 45, first noticed an eruption on the palate in July 1932, and in October of the same year swelling of the upper lip became apparent. Since then this had increased only slightly, although it fluctuates from day to day. There is no history of febrile attacks connected with the increased swelling and pain in the lip; there have been no subjective disturbances, and the patient is otherwise well, so far as she knows.

At the junction of the hard and the soft palate there is a group of fairly soft papules extending symmetrically outward from the median line and forward on each side in a sulcus about 0.5 cm. lateral to the median line. The major part of this group is about 2 cm. wide and 1.5 cm. in the anteroposterior measurement. Some of the papules are round; the larger ones are elongated. The upper lip shows moderate enlargement and is not hard. The skin on the left side of the lip is somewhat redder than that on the rest of the face.

The Wassermann, Kahn and Pirquet tests were negative. Clinical and roentgenologic examinations of the chest revealed no abnormalities.

Examination of the blood showed: erythrocytes, 3,900,000; leukocytes, 6,150; hemoglobin (Sahli), 67 per cent; polymorphonuclear neutrophils, 71.5 per cent; lymphocytes, 28.5 per cent.

Biopsy on one of the small papules from the palate showed a thickened, edematous epithelium and marked edema of the corium, with an infiltrate of connective tissue cells which was so thick at the center of the papule that the fibers could hardly be seen. The connective tissue fibers throughout were much swollen. Elastic tissue fibers stained by the Hart modification of the Weigert method were very few and fine, chiefly in the less affected papillary layer.

DISCUSSION

DR. A. H. CONRAD, St. Louis: I noticed a geographic tongue. Has that any connection with the diagnosis given?

DR. A. W. STILLIANS, Chicago: I am wondering what is the connection between the condition of the palate and that of the lip. I do not consider the geographic tongue as of any importance.

TELANGIECTASIA (HEREDITARY, HEMORRHAGIC)? OSLER'S "RECURRING EPISTAXIS WITH MULTIPLE TELANGIECTASES OF THE SKIN AND MUCOUS MEMBRANES"? Presented by DR. THEODORE K. LAWLESS, Chicago (by invitation).

This woman, aged 27, presents an eruption which involves principally the face, ears, nose, tongue and trunk. She states that some of the lesions were present at birth but that recently there has been a marked increase in the number and size of the lesions. She gives a history of frequent attacks of epistaxis since infancy.

The family history reveals that the paternal and maternal grandparents died of carcinoma. The father when a child had spinal meningitis which produced complete loss of speech and hearing. The mother at the age of 12 had "brain fever," with loss of speech and almost complete loss of hearing, and she has many brown macules on the chest. One sister had a nevus on her cheek. The patient had the usual diseases of childhood and underwent abdominal operations in 1925 and 1933. She has never been pregnant.

There are now countless red lesions, papules and tumors, varying from the size of a pinpoint to 3 cm. in diameter as well as angiomas and telangiectatic vessels of various dimensions, well distributed but particularly numerous on the face, ears, tongue and inner nares. These lesions are noninflammatory and do not fade on pressure.

The Wassermann and Kahn reactions were negative. The blood cell count and hemoglobin were normal. The blood pressure was 110 systolic and 80 diastolic. The basal metabolic rate was —18.6.

DISCUSSION

DR. S. W. BECKER, Chicago: I observed a case several years ago which was somewhat similar to this. I question the diagnosis of Osler's disease because his cases were distinctly familial, and there is no history so far as I could learn of the presence of a similar condition in this family. However, a familial condition has to start somewhere, and it will be interesting to see if the same malady occurs in this woman's children. My patient was an unmarried man. There is always a possibility of such a condition being atavistic.

DR. JOHN F. MADDEN, St. Paul: I think that the condition is hereditary hemorrhagic telangiectasia. The patient states that her father has had frequent nosebleeds for years. Many patients with hereditary hemorrhagic telangiectasia have telangiectasia without hemorrhage; or hemorrhage without demonstrable telangiectasia, for many years before the other sign or symptom is noticed. Of course, the typical patient has both telangiectasia and hemorrhage.

MYCOSIS FUNGOIDES. Presented by DR. E. P. ZEISLER, Chicago.

This man, veterinary surgeon aged 65, states that the onset of his trouble was in 1917. In 1918 a diagnosis of toxic dermatitis was made at the Mayo Clinic, and the tonsils and several teeth were removed.

He was first seen by me in 1920, with annular and gyrate, plaquelike lesions of the trunk which suggested either parapsoriasis or the premycotic stage of mycosis fungoides. The lesions cleared up under arsenic and roentgenotherapy, and the patient remained well for three years. Since that time new lesions, including definite tumors, have appeared at varying times and have been kept under control with arsenic and roentgenotherapy. Splenic extract was used for a time, but was without effect. Arsenic has been given to the limit of tolerance, in the form of a solution of potassium arsenite or pills. The general health has not been affected except for marked pruritus.

On Dec. 7, 1933, a blood count showed: erythrocytes, 5,620,000; leukocytes, 6,450; hemoglobin, 90 per cent; polymorphonuclear neutrophils, 74 per cent;

lymphocytes, 19 per cent; monocytes, 3 per cent; eosinophils, 3 per cent, and basophils, 1 per cent.

A biopsy specimen is presented. The histologic picture is that of mycosis fungoides.

The patient is presented because of the remarkable period (seventeen years) that the disease has lasted, and particular attention is called to a tumor of the right eyelid.

MYCOSIS FUNGOIDES. Presented by DR. S. W. BECKER, Chicago.

This patient was presented at the December 1933 meeting of the Chicago Dermatological Society, and the history was published in the transactions of that society (ARCH. DERMAT. & SYPH. **30**:135 [July] 1934.

DISCUSSION

DR. S. W. BECKER, Chicago: I have had a chance to observe this man from the inception of the trouble. The condition was clinically typical lichen spinulosus that might have been due to arsenic or bismuth. I suspected a leukocytic dyscrasia, for the only thing that would control the condition was roentgenotherapy. A small tumor finally developed after about two years of observation, and the diagnosis of mycosis fungoides was made histologically.

DR. WILLIAM ALLEN PUSEY, Chicago: Such cases are extremely interesting and well worth seeing, but the members are in general agreement and it is of no importance that they are not discussed *in extenso*.

A CASE FOR DIAGNOSIS (PARAKERATOSIS VARIEGATA WITH CHANGES DUE TO ARSENIC?). Presented by DR. F. E. SENEAR and DR. HERBERT RATTNER, Chicago.

This patient was presented first at the December 1926 meeting of the Chicago Dermatological Society and again at the annual meeting of that society in January 1927. The case is recorded in the ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY (**15**:739 [June] 1927; **16**:89 [July] 1927).

The patient reported to the University of Illinois one week ago. The eruption remains essentially the same as when he was first presented but has spread to involve the entire body—trunk, extremities, face, neck and ears. The telangiectasia has become much more prominent, giving the skin on first glance the appearance of chronic radiodermatitis.

Biopsy revealed a thin scale with islands of parakeratosis. In some areas the epidermis showed acanthosis with widening and fusion of the rete pegs, while in others there were atrophy and thinning so that only two or three cell layers remained. There was marked intracellular and intercellular edema, most pronounced in the basal layer. The granular layer was discontinuous. In the atrophic areas the basal layer was disorganized, and there was no sharp demarcation between the epidermis and the corium. The upper part of the corium was edematous and very vascular, and all of the vessels were widely dilated. Throughout this zone there was a diffuse infiltration composed of lymphocytes and histiocytes. Thin mantles of similar infiltration extended down about the deeper vessels. In the upper part of the corium the collagen fibers were markedly thinned, while the elastic fibers were absent.

DISCUSSION

DR. M. H. EBERT, Chicago: I had an opportunity to study this man on several occasions, and when I saw the patient whom Dr. Ormsby and I presented today with a diagnosis of parakeratosis I at once thought of this man. The condition impressed me as the same sort of disease, and I was interested in seeing the biopsy specimen and comparing the two. This man's disease has progressed further, the keratotic scaling is more marked and there is more

telangiectasia. Under the microscope there are more pronounced changes than in the case of the patient whom we presented. The areas of edema in the rete mucosa are more marked in places, and while I could not find areas of telangiectasia I presume they would be observable on further study.

DR. OLIVER S. ORMSBY, Chicago: Dr. Montgomery, who is now in California, made the test for arsenic, using the Osborne technic, and he found arsenic in the scales.

ARGYRIA. Presented by DR. THEODORE CORNBLEET and DR. E. R. PACE.

This patient was presented at the November 1933 meeting of the Chicago Dermatological Society, and the history was published in the transactions of that society (ARCH. DERMAT. & SYPH. **29**:775 [May] 1934).

He is presented at this time to demonstrate bleaching by the injection of sodium thiosulphate and potassium ferrocyanide, according to the technic of Dr. Stillians and Dr. Lawless. An area on the right trochanteric region was inoculated once in November 1933, and a nearby wheal was inoculated today, with successful bleaching of the site.

MELANOMA. Presented by DR. F. E. SENEAR and DR. M. R. CARO.

This white man, aged 28, states that he had no lesions on the face during early childhood, and this is substantiated by photographs. When he was 7 years of age, as a prank, some unidentified black powder was blown over his face, without, however, injuring the skin. All of the powder was removed except that over the site of an area of discoloration now present, which has remained stationary in size since that time. At 9 years of age he lighted a shotgun shell and sustained a powder burn on the face, but this produced no change in the appearance of the lesion. At 11 years of age he was bitten on the right eyebrow by a mosquito. This was followed by an abscess, which drained for a long time and resulted in the formation of a soft, cystlike tumor at this site which remained stationary in size until the past year, when it enlarged slightly. It was removed in October 1933 and was found to be a melanoma. Radium therapy was then given locally.

There is now a palm-sized area of bluish discoloration on the right side of the forehead, circumscribed above but infiltrating at the sides and below to involve the right upper eyelid and the medial portion of the lower lid. At the eyebrow is a scar left by the excision. Scattered throughout the area of discoloration are several slightly elevated papules the size of a pinhead, some darker than the base and others depigmented. These have been present for from three to four years without much change in their appearance.

Histologic examination of a biopsy specimen including one of these depigmented papules (hemalum-eosin stain) showed the epidermis to be normal. In the upper part of the corium, extending nearly to the epidermis, was a small papule which was pierced by a hair and a sebaceous gland. The papule was composed of densely packed, wavy, pale-staining fibers, running in parallel and intertwining strands, and it also contained a few pigmented cells and many nerve fibers. Below and to one side of this papule there was deeper extension of similar tissue. In the surrounding corium there were many nerve fibers, several dense pigmented masses in which the pigment lay within cells and fibers and many scattered pigmented cells.

DISCUSSION

DR. S. W. BECKER, Chicago: I think that the condition is blue nevus which might be mistaken for melanoma. There is a history of having some powder thrown on the forehead, but I think that it had nothing to do with the lesion. The so-called mongolian spot gives this same appearance. It is interesting that since the lesion appeared at the age of 7 years it has not extended but small tumors have appeared. The small tumors are not malignant, and Masson

would say that they are schwannomas. He believes that pigment-forming cells have something to do with the sheath of Schwann and that they show this picture when benign neoplasms develop from them.

DR. RUBEN NOMLAND, Chicago: Does Dr. Becker think that the cells are like those of a blue nevus or of a mongolian spot?

DR. S. W. BECKER, Chicago: The pigmented cells are the same as those found in blue nevi.

DR. F. E. SENEAR, Chicago: A small tumor of the periorbital region developed; this was excised, and a diagnosis of melanoma was made by Dr. Jaffé at the University of Illinois. The history of powder being thrown in the patient's face and of another explosion at one time has led the family to believe that the condition is an artificial tattoo. We do not believe this to be the case but are particularly interested in the small white lesions, and in order to rule out the possibility of tattoo a biopsy was made, which revealed the changes described.

DERMATITIS EXFOLIATIVA NEONATORUM (LEINER'S DISEASE)? Presented by DR. THEODORE CORNBLEET, Chicago.

This Negro infant was born prematurely of a tertipara on Jan. 4, 1934, by normal delivery. At birth he weighed 4 pounds and 6 ounces (1,984.47 Gm.) and presented exfoliation over the lower extremities and the tips of the fingers. On the next day the child was spastic, and coarse tremors of the upper extremity were noted. The Wassermann reaction was negative. Five days after birth many pinhead-sized vesicles developed on the forehead and a few pustules under the chin. The vesicles extended to the shoulders, chest and abdomen. Twelve days after birth the child was described as having vesicular dermatitis with almost universal exfoliation, the palms, soles and lower portion of the face being spared. There was no bright erythroderma underlying the exfoliation.

DISCUSSION

DR. O. J. CAMERON, Omaha: Depending on the degree of involvement, it is sometimes difficult to judge whether one is dealing with Leiner's erythrodermia desquamativa or Ritter's dermatitis exfoliativa neonatorum. Guy and Cohen (ARCH. DERMAT. & SYPH. **19**:425 [March] 1929) reported a case of Ritter's disease with autopsy, one of the most significant observations being enlargement of the suprarenal glands and massive hemorrhage into one of them. A case of Ritter's disease has been observed at the University of Nebraska Hospital in which massive hemorrhage into the suprarenal glands was found at autopsy.

DR. EVERETT S. LAIN, Oklahoma City: Recently Dr. Michael in his clinic showed a patient with a condition which, in many respects, was similar to this, and I think that he should discuss the subject.

DR. JEFFREY C. MICHAEL, Houston, Texas: The case of mine mentioned by Dr. Lain was a classic example of Ritter's disease. In the present case the condition does not seem to fit into the picture of that disease. Neither does it suggest Leiner's disease, which shows much more marked inflammatory features. I have seen only one example that I felt was Leiner's disease and in that case the eruption cleared promptly after the injection of the mother's blood, which was the treatment suggested at the time by Leiner, I believe.

DR. O. H. FOERSTER, Milwaukee: The epitrichial layer may be concerned in the pathogenesis of this condition. The absence of marked symptoms of infiltration, I think, gives support to that suggestion.

DR. THEODORE CORNBLEET, Chicago: The patient is a premature child, now about 18 days old. The condition seemed to be subsiding rather rapidly when I first saw the infant about ten days ago. There was some erythroderma underlying the infiltration. There were no large bullae, which made me reject the possibility of its being merely a pyogenic infection, which I had in mind. There was seborrhea behind the ears, which is supposed to accompany Leiner's disease. I do not think that the epitrichial exfoliation is responsible for the dermatitis seen at that time.

NECROBIOSIS LIPOIDICA DIABETICORUM. Presented by DR. E. P. ZEISLER and DR. M. R. CARO, Chicago.

This patient was presented at the clinical session of the American Dermatological Association in June 1933, and the history was published in the transactions of that association (ARCH. DERMAT. & SYPH. **29**:167 [Jan.] 1934).

DISCUSSION

DR. C. C. DENNIE, Kansas City, Mo.: I saw this patient last June, and within two weeks after I got home I saw a girl, aged 12, with severe diabetes who suddenly presented the same picture. The only way in which the lesions differed from the ones seen in this case was that they were more sclerotic, had a different color, were sharper in outline and ulcerated through the entire skin.

DR. H. E. MICHELSON, Minneapolis: I have been fortunate enough to observe a patient with this condition and have given the case considerable attention. As is well known, the disease was first observed by Oppenheim and later discussed and given the present name by Urbach. Most of the patients have severe diabetes. The differentiation from xanthoma diabeticorum is made both clinically and histologically. In necrobiosis lipoidica diabeticorum there are no foam or giant cells. The precise fat which constitutes the lesion has not been determined. It undoubtedly is a mixture. One may speculate about the pathogenesis, and since it is not closely allied to xanthoma my associates and I thought that it must be related to some other common condition found in diabetes. We, therefore, studied sections taken at the periphery of diabetic gangrene and found a marked similarity. At the present time it is our opinion that necrobiosis lipoidica diabeticorum is an abortive form of diabetic gangrene.

DR. M. R. CARO, Chicago: Three weeks after seeing this patient we were fortunate in seeing a woman, aged 72, who had mild diabetes mellitus and pemphigus vulgaris and a single patch of necrobiosis lipoidica diabeticorum on the outer surface of the left foot. The histologic changes in this patch were the same as in Urbach's case. The patient died of pemphigus, and a postmortem examination was performed, the results of which will be incorporated in a future report.

TULAREMIA? SPOROTRICHOSIS? Presented by DR. THEODORE CORNBLEET and DR. E. R. PACE, Chicago.

This Negress, aged 40, noted a papule on the right little finger three months ago. She stated that she squeezed some pus from the papule and it healed in about two weeks, but shortly afterward new lesions appeared, one at a time, on the hand, wrist and forearm.

She entered the hospital two weeks ago with six nodules which were discrete and arranged in a linear fashion. They were purple, firm and deeply indurated, and there was fluctuation in one. There was slight tenderness but no pain. The epitrochlear and axillary glands on the affected side were enlarged and tender. There was no history of contact with rabbits, nor had the patient been ill recently.

Pus from the site of biopsy was cultured nine days ago but as yet shows nothing. The patient left the hospital at that time, which prevented testing her blood for agglutinins for Sporotrichum.

Biopsy revealed caseous tuberculosis.

The state laboratory reported the blood to give a positive agglutination reaction for Bacillus tularensis in a dilution of 1:1,280.

DISCUSSION

DR. RICHARD L. SUTTON, Kansas City, Mo.: I am interested in this case because one occasionally encounters such cases. The condition is probably tularemia. Formerly I saw a good deal of sporotrichosis, but I have seen none in the past two years. I do see cases of tularemia, particularly in consultation,

and these lesions are supposed to result from infection via rabbit blood, but they are not necessarily due to that. I saw one that was due to the bite of a chicken. It is easy to send blood to the laboratory and have it tested for agglutinins for B. tularensis. I think that this patient has tularemia of the sporotrichotic type.

DR. H. R. FOERSTER, Milwaukee: Although the history is suggestive of sporotrichosis and some of the lesions may fit into the clinical picture, I do not believe that there is sufficient evidence to justify that diagnosis. The lesion at the apparent site of infection developed following trauma, appears to have been insignificant in its manifestations and healed spontaneously, which points more to tularemia than to sporotrichosis. The absence of lymphangitis, the presence of regional adenopathy and the spontaneous healing of the cutaneous lesions are also against sporotrichosis and in favor of tularemia. Might the condition not be low grade tularemia?

DR. RICHARD S. WEISS, St. Louis: I do not claim that the condition is sporotrichosis, but I protest against a diagnosis of tularemia for a patient who is not ill. The patients with tularemia that I have seen had fever, presented a course typical of typhoid and, even when tularemia was of the sporotrichoid type were quite ill. However, there may be a mild type of the disease with which I am unfamiliar.

DR. THEODORE CORNBLEET, Chicago: We thought of inoculation tuberculosis of the skin, sporotrichosis and tularemia, but we are not yet sure what the patient has. We do not think it is tularemia because she has not been sufficiently ill. She has shown no symptoms during the course of the disease. Today, however, we received a report from the state laboratory that the serum agglutination test gave a positive reaction for B. tularensis in a dilution of 1:1,280, which is considered evidence of tularemia. Those who looked at the tissue section saw the typical structure of tuberculosis, and we are not sure that the patient does not have inoculation tuberculosis of the skin.

DR. JOHN F. MADDEN, St. Paul: Is it not true that if a patient once gives a positive agglutination reaction for tularemia the reaction remains permanently positive? This patient may have had tularemia at some time in the past and the present positive reaction may be the result of the previous disease.

PITYRIASIS LICHENOIDES ET VARIOLIFORMIS (ACUTA). Presented by DR. HERBERT RATTNER.

This Negress, aged 49, from the Mandel Clinic, presents a generalized eruption of four or five months' duration. She was first seen in May 1933 because of aortitis. The Wassermann reaction was 1 plus, and the Kahn reaction, 4 plus. She has had four normal pregnancies and no miscarriages. She was treated with iodides and mercury by mouth. She has diabetes, now under control.

On her return to the clinic two months ago she presented a generalized eruption of three months' duration, with lesions distributed symmetrically on the trunk, extremities and sides of the face. The oral mucosa, the flush area of the face and the palms and soles were not involved. The eruption was polymorphous, consisting of numerous vesicles, some pustules, scaly papules and blood-crusted lesions. They were discrete and distinctly inflammatory, each lesion being surrounded by a hyperemic zone. The lesions first appeared on the arms and legs, apparently in crops, and only the new lesions were pruritic. The Wassermann as well as the Kahn reaction of the blood was 4 plus. There were no systemic symptoms and no adenopathy.

The eruption now consists of pea-sized, lichenoid, rounded papules, deeply pigmented macules, and a few crusted lesions arranged symmetrically on the forehead, sides of the face, neck, trunk and extremities. The palms, soles and mucous membranes are free from lesions. Four weeks ago the Wassermann reaction of the blood was negative and the Kahn reaction, 4 plus. She has had no treatment.

Biopsy on an early lesion (Dr. Caro) showed a central crust and at the sides a thin, non-nucleated scale. The epidermis showed slight intercellular and intracellular edema and beneath the crust regeneration from the sides. In the upper part of the corium the blood vessels were surrounded by thin mantles of infiltration, composed of lymphocytes, macrophages and neutrophils, with a few eosinophils. Thicker mantles of similar infiltration were noted about the sweat glands and deeper vessels.

DISCUSSION

DR. LOUIS A. BRUNSTING, Rochester, Minn.: How does Dr. Rattner distinguish this condition from bromoderma or iododerma. From the history, the patient was receiving iodide therapy when the eruption developed.

DR. E. A. OLIVER, Chicago: I looked at this patient first without knowing the history and made a diagnosis of papulonecrotic tuberculid. The histologic observations are not incompatible with that diagnosis. I was surprised to learn that the patient was presented with the diagnosis of pityriasis lichenoides et varioliformis acuta. I do not think that she presents the picture of that disease today. In the average case that disease looks much like maculopapular syphilis in which among the maculopapules are seen vesicles, hemorrhagic crusted lesions and superficial scarring. Dr. Senear tells me that when he first saw this patient she did present that picture, with vesicles and hemorrhagic crusting. That being the case, I readily accept the diagnosis. The picture today is confusing.

DR. F. E. SENEAR, Chicago: When I first saw the patient with Dr. Rattner several weeks ago she had the lesions that I should expect to find in pityriasis lichenoides et varioliformis acuta. The first patient whom Dr. Oliver and I saw showed a much more extensive and much more severe form of the disease. The rapid course of the disease and the clinical appearance of the lesions are not compatible with the diagnosis of papulonecrotic tuberculid. Today the type of scarring is suggestive of that condition, but the histologic picture is not at all typical.

DR. HERBERT RATTNER, Chicago: The patient received iodides for three weeks, and the eruption developed three months after the treatment was discontinued. It is clearing up rapidly. She formerly presented vesicles and blood-crusted lesions, but the eruption was predominantly papular. The polymorphous character, the distribution of the lesions, which leave pigmented relics, and the general course seem to fit in well with the descriptions usually given of pityriasis lichenoides et varioliformis.

PLICA NEUROPATHICA. Presented by DR. F. E. SENEAR, Chicago.

This infant, aged 20 months, presents a disorder of the hair. The mother states that about the middle of October 1933 she noticed that the hair was becoming very dry and was forming "snarls," which came out with combing. Since that time the matting of the hair has progressed rapidly, from twelve to fifteen places developing within a week. When some of these felted strands of hair were clipped, it was found that the individual hairs were curled and interlaced but showed no marked change, although trichorrhexis nodosa was noticed in some of them.

DISCUSSION

DR. RICHARD L. SUTTON, Kansas City, Mo.: This is an extremely interesting case, and I wish to make three suggestions: first, that a biopsy specimen from the center of one patch be examined to determine what the character and course of the hair follicles show; second, that histologic study of the hair on cross-section be made; third, that the child's head be kept shaved for several weeks and the effect observed.

DR. F. E. SENEAR, Chicago: I brought this child in because I had never seen a similar condition. Several persons have asked about the possibility of

this being an artefact. I can say only that by the observation of the child by the parents and by the nurse during the night that element has been eliminated. I have not as yet made studies on the hair from the standpoint of cross-section, but under the microscope nothing is apparent in the hair as a whole. I appreciate Dr. Sutton's suggestion regarding a biopsy, but I fear that it will be impossible to obtain a specimen.

MINNESOTA DERMATOLOGICAL SOCIETY

L. H. WINER, M.D., *Secretary*

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D. D. TURNACLIFF, M.D., *President, Presiding*

CUTIS VERTICIS GYRATA (CONGENITAL ANOMALY TYPE). Presented by DR. JOHN F. MADDEN, St. Paul.

This schoolboy, aged 6 years, has had a large mass on the anterior half of the right side of the scalp which has gradually increased in size since birth. The lesion was smooth, not elevated, slate blue and hairless at birth. He was born at full term by normal delivery, and was breast fed. He has had no convulsions or other unusual conditions of childhood. The mass on the head has grown rapidly during the past year, and this has been accompanied by glandular enlargement in the submaxillary and postauricular regions.

There are no abnormalities in the family history.

The lesion consists of parallel folds running anteroposteriorly and covering the greater part of the anterior half of the right side of the scalp. The folds are thicker in the middle of the lesion and taper off at the margins. There is a walnut-sized knob extending anteriorly from the middle fold. The entire lesion is painless. It is dirty white with a tinge of blue to deep bluish red. The deepest color is in the projecting knob. The lesion has the consistency of a solid rubber ball, but the anterior knob is harder than the rest of the lesion. The right postauricular and submaxillary lymph glands vary in size from that of a bean to that of an olive and are firm and painless. The physical examination otherwise gave negative results.

Roentgenograms of the head taken on Dec. 21 and 23, 1933, at various angles, showed no evidence of any tumor or involvement of the bone.

The urine was normal. Examination of the blood on December 22 showed: red cells, 4,460,000; hemoglobin, 93 per cent, and white cells, 13,200, of which 57 per cent were polymorphonuclears, 40 per cent lymphocytes, 1 per cent monocytes, 1 per cent eosinophils and 1 per cent basophils. The bleeding time was one and one-half minutes; the clotting time, four and one-half minutes.

The biopsy specimen was seen by Dr. E. T. Bell, who said that the picture was that of an inflammatory fibrosis. The specimen is shown here.

DISCUSSION

DR. H. E. MICHELSON, Minneapolis: This case has been under discussion for several weeks. When I first saw the slide I thought of Recklinghausen's disease, but tonight I am inclined to think that the lesion is showing signs of a malignant change, and it is my feeling that sarcomatous change is taking place.

DR. HAMILTON MONTGOMERY, Rochester: The histologic picture is striking. It definitely rules out endothelioma capitis (cylindroma or nevoid cylindric epithelioma). There are groups of cells apparently in close relation to or arising from the walls of the blood vessels. In addition, there are cells arranged in whorl formation suggestive of the histologic picture described by Duemling (Cutaneous Neuroma: Report of an Unusual Case Simulating Keloid, ARCH. DERMAT. &

SYPH. **19**:226 [Feb.] 1929). The histopathologic picture is not that of the ordinary Recklinghausen's disease. Both that condition and neuroma can at times show malignant or sarcomatous changes. The bluish area presented clinically by this patient I am inclined to interpret, however, as the result of vascular stasis due to proliferative changes interfering with the circulation to this area rather than as a malignant change of the tumor. The case deserves detailed histopathologic study with special stains before a final diagnosis is made, and, if possible, further biopsy specimens from early as well as later lesions should be taken. The condition is not a ganglioneuroma, and no ganglion cells were present in the sections that were shown.

DR. JOHN F. MADDEN, St. Paul: My associates and I were going to remove the entire lesion surgically, drill holes through the outer table of the skull and graft skin if necessary. Has any one any other treatment to suggest?

PSORIASIS ARTHROPATHICA. Presented by DR. JOHN F. MADDEN, St. Paul.

This man, aged 45, a laborer, states that in 1920 he had acute gonorrheal urethritis. During the course of this infection pain, redness, swelling and tenderness of the ankles, knees and elbows developed. The disease was diagnosed as acute rheumatic fever. Along with this, he had crusted lesions on the toes and hands. The cutaneous lesions remained, but the manifestations in the joints subsided. In 1925 the patient again had involvement of the joints, and new lesions appeared on the hands, feet, knees and elbows. The skin eruption has remained about the same for several years except that new lesions have appeared on the face.

The patient presents deforming arthritis of the ankles, the knees and the small joints of the hands and feet. The skin is atrophic and glazed over the knees and hands. There is a generalized eruption which is more marked on the elbows, hands, knees, feet and face. This consists of various-sized patches and plaques. The lesions have an erythematous base which is capped with a white or yellow, greasy or dry, thick scale. The nails of all of the fingers and toes are markedly thickened, and all show subungual keratoses.

The genito-urinary department of the hospital has been unable to demonstrate gonococci at any time. It has not been possible to obtain material for biopsy.

DISCUSSION

DR. PAUL O'LEARY, Rochester: I agree with the diagnosis of psoriasis arthropathica. The essential feature of this type of arthritis is the original involvement of the terminal phalanges, a characteristic which I am told is not found in other forms of arthritis. This patient's psoriasis is approaching complete generalization; the deforming arthritis is a part of this syndrome. Ultraviolet irradiation and treatment with tar, as suggested by Goeckerman, augmented by vaccine therapy and baking, help to produce remissions in these patients.

DR. H. G. IRVINE, Minneapolis: This patient was shown by Dr. Turnacliff and me, and at that time there was considerable discussion as to whether he had syphilis or psoriasis because the lesions had cleared up under bismuth medication. Several of the members had seen him before that, presenting psoriasis. He had had psoriasis without doubt, but at the time we showed him the areas were almost clear.

PSORIASIS. Presented by DR. JOHN F. MADDEN, St. Paul.

This woman, aged 27, a housewife, noted an eruption on the distal part of the forearms about Jan. 7, 1934. The eruption became generalized in about two weeks. The lesions do not itch and have been dry throughout their course. The eruption consists of split pea-sized papules capped with a thick, dry, white scale. Bleeding points appear when the scale is removed. The patient accidentally scratched herself on the forearm, and lesions appeared in the scratch marks.

The Wassermann reaction of the blood was negative.

A biopsy specimen is shown.

DISCUSSION

DR. PAUL O'LEARY, Rochester: The appearance of the psoriatic papules in the linear scratch marks is known as the phenomenon of Koebner. He believes it to be one of the factors supporting the contention that psoriasis is an infectious disease.

ONYCHOGRYPOSIS. Presented by DR. JOHN F. MADDEN, St. Paul.

This woman, aged 54, a housewife, states that for the past eight years she has had arthritis involving the joints of the hands and feet. For the past two years she has not cut her nails because the parts have been swollen and sensitive. Because of this she also has refrained from washing her hands and feet. The patient has remained in bed most of the past seven years and has not washed her hands and feet for over two years. The nails are thickened and curved. Some nails are over an inch in length. There is subungual keratosis under each nail. The skin of the palms and soles is thickened and covered with a heavy scale. The dorsa of the hands and feet have a dry, atrophic appearance and are covered by an abundant scale which peels off in large flakes.

DISCUSSION

DR. PAUL O'LEARY, Rochester: I should like to discuss this disease under the name of rupial impetigo. During the past month I have had under observation a girl of 16 years with an identical process. She also has extreme arthritis. Practically all of her joints are "solid." The joint fixation has made it difficult for her parents to cleanse her hands and feet properly since the fingers and toes are immobile. The heaped-up, inspissated and rupial crusts are readily removed after applying a wet dressing for forty-eight hours, and the underlying skin is found to be but superficially involved. The clawlike finger-nails come off with the débris, and new ones readily form. The use of an antiseptic ointment after removal of the crusts has resulted in rapid healing in my case.

DR. HAMILTON MONTGOMERY, Rochester: Histologic section in Dr. O'Leary's case showed layers of completely keratinized corneal cells, every fifth to eighth layer being separated by a thin layer composed of masses of staphylococci. The corneal material was not parakeratotic as is usually seen in keratoderma, and no intracellular gram-negative diplococci were present.

DR. E. C. GAGER, St. Paul: I do not see this condition in females frequently, although I have run across one or two cases. The lesions on the feet resemble those of blennorrhagia. I see no reason why a coccic infection might not produce the same picture. Possibly the history in this case would be informative, because keratoderma blennorrhagica begins as a pustular eruption and only after a time produces a scale or a complete cast. If it has been present only for six months and the lesions appear to be large plaques, that is an indication that one is not dealing with true gonorrheal blennorrhagia.

DR. PAUL O'LEARY, Rochester: The possibility of keratoderma blennorrhagica was considered in my case also. The vaginal smears were negative for gonococci. Histopathologic study of one of the masses showed that the lesions were not keratotic but were heaped-up layers of débris with many bacteria of all sorts between the layers, which seemed to justify the elimination of the possibility of keratoderma blennorrhagica. The striking feature in both of these cases is the extreme degree of arthritis, involving practically every joint and thus resulting in an unkemptness which led to the development of these lesions.

DR. JOHN F. MADDEN, St. Paul: The history in this case is practically the same as that in the case that Dr. O'Leary mentioned except that the eruption is of longer duration. This is the first time since the patient has been in the hospital that one has been able to persuade her to permit treatment of her feet or hands.

DERMATITIS HERPETIFORMIS. Presented by DR. JOHN F. MADDEN, St. Paul.

About Nov. 1, 1933, the mother of R. S., a boy aged 22 months, noticed some small pustular lesions on his face. The eruption has increased rapidly until most of the skin surface is involved. The lesions have appeared in crops and are very itchy. There has never seemed to be spreading from an existing lesion to normal skin. The eruption is cyclic. The skin shows crops of grouped, tense, pinhead-sized to pea-sized vesicles which are roughly symmetrical. Other parts of the skin show the residual erythema and pigmentation of previous lesions. The eruption can be markedly aggravated in twenty-four hours by three doses of 1 minim (0.06 cc.) of a saturated solution of potassium iodide.

The blood shows that 25 per cent of the leukocytes are eosinophils. The vesicles themselves show 40 per cent eosinophils.

DISCUSSION

DR. H. G. IRVINE, Minneapolis: I agree that dermatitis herpetiformis is one diagnosis that should be considered in this case, but I cannot see anything which makes it possible to pin the possibilities down to that. The condition is exceedingly rare in a child of the age of the patient. All the cases that I have seen in children have begun at the age of 3 or 4 years with an itching dermatitis and after a period of years have developed to the stage at which one could make a definite diagnosis of dermatitis herpetiformis. On the other hand, I have seen conditions diagnosed by two of my colleagues as pemphigus which cleared up after the removal of tonsils. This child has a history of sore throat. I think that there should be further study, but I am inclined to list the condition as a toxic eruption until it is more characteristic than at present.

DR. JOHN F. MADDEN, St. Paul: The patient has been given no iodides or bromides except what he has received here. He does not use salt. He has been under observation for two months and has been given iodides only during the past two weeks.

DR. PAUL O'LEARY, Rochester: The weight of clinical evidence is in favor of dermatitis herpetiformis. However, the possibility of iododerma must be eliminated.

DR. H. E. MICHELSON, Minneapolis: My associates and I have been much interested in dermatitis herpetiformis for the past few months, and I have outlined a problem for Dr. Madden on this disease. I believe that this condition is diagnosed too often in this country. The work on sensitization to halogens should be repeated as well as that on the value of eosinophilia as a diagnostic aid.

DR. A. L. WELSH, Rochester (by invitation): For the past fifteen months I have been working from the bacteriologic standpoint on pemphigus and several other bullous dermatoses which resemble it. In the past few months I have discovered what I think will eventuate in a specific test for pemphigus. This test would be applicable in precisely this type of case. I am hoping in the near future to get blood from patients with eruptions such as this, and with other bullous dermatoses, on which to make the test. If the procedure continues to be as clear-cut with these serums as it has been with the serums on which it has been tried, this will be a relatively rapid and exact differential test. I should be pleased to receive sterile blood from patients with suspected pemphigus or other bullous dermatoses for this test.

DR. L. H. WINER, Minneapolis: Many European clinicians do not separate Duhring's disease from chronic pemphigus. I have seen one patient with an eruption of this nature who was benefited by the removal of abscessed teeth and by tonsillectomy.

DR. FRANCIS W. LYNCH, St. Paul (by invitation): If one accepts the essential identity of dermatitis herpetiformis and herpes gestationis, one might recall Howard's mention of cutaneous lesions in early infancy in children born of mothers suffering from this condition (Herpes Gestationis: Report of a Case, ARCH.

DERMAT. & SYPH. **28**:782 [Dec.] 1933). There would then be no objection to a diagnosis of dermatitis herpetiformis because of the age of this child. I have been unable to verify Howard's reference to Milton's cases in order to determine whether the eruption was actually present at the time of birth (*J. Cutan. Med. & Dis. Skin.* **1**:311, 1867-1868).

DR. H. G. IRVINE, Minneapolis: Dermatitis herpetiformis may be improved by removal of teeth and tonsils, but I know of no cases in which it has cleared and stayed cleared. In cases of this type I feel that a diagnosis of dermatitis herpetiformis cannot be made except when more symptoms are present.

DR. A. L. WELSH, Rochester (by invitation): I do not think that the lack of improvement after the removal of a focus such as tonsils in dermatitis herpetiformis, or in any disease, can be interpreted as evidence that the disease is not the result of focal infection. It has been shown by many workers that if one structure becomes a focus of infection, secondary foci soon appear, and similar organisms in lymphoid tissue and on the mucous surfaces soon acquire the same specific localizing ability as those in the primary focus. Hence, one can expect favorable results following removal of the primary focus only when this focus is removed before the development of secondary foci. This has been clearly shown in many diseases, particularly in arthritis.

FIXED ERUPTION DUE TO BISMUTH. Presented by DR. S. E. SWEITZER, Minneapolis.

This white man, aged 32, was admitted to the Minneapolis General Hospital on Jan. 12, 1934, with macular hyperpigmented areas over the body which had been present for about five months. Prior to the onset of the present complaint the patient had been treated with neoarsphenamine and bismuth for secondary syphilis, the first year of treatment being at the Minneapolis General Hospital in 1931. There was no reaction following this medication, and the Wassermann reaction of the blood was negative. In 1932 the patient was treated by a private physician, and pruritic erythematous areas developed on the arms and fingers which became vesicular and appeared as dermatitis venenata following intravenous injection of neoarsphenamine. The Wassermann reactions of the blood and spinal fluid at that time were negative. In September 1933 the patient returned to a penal institution in Minneapolis and was found to have a repeatedly positive Wassermann reaction. Because of the history of reaction to neoarsphenamine, bismuth subsalicylate in oil was used in treatment. However, the patient noted after each injection urticaria-like lesions and severe pruritus of the fingers, hands and arms and also headaches which persisted for two or three days. After several treatments had been given, the patient noticed soreness of the gums and slate-blue pigmented areas over the body. These were present at the time of his admission to the hospital.

There is no history of drug ingestion. The physical examination revealed nothing abnormal except unequal pupils, bluish discoloration forming a line at the borders of the gums and oval to round, sharply demarcated slate-blue hyperpigmented areas varying in size from that of a split pea to that of a quarter over the extremities, neck and groins. The cutaneous lesions showed no surface change or induration and were not tender to pressure. The spinal fluid was normal; the Wassermann and Kahn reactions of the blood were 4 plus. Patch tests with neoarsphenamine and bismuth were negative. A biopsy specimen is shown.

A CASE FOR DIAGNOSIS. Presented by DR. JOHN F. MADDEN.

This man, aged 40, Mexican, had a penile chancre many years ago. The lesion was treated locally. He received five injections intravenously in 1924. He was next treated in May 1933 with neoarsphenamine and bismuth salicylate in oil. The second injection of neoarsphenamine was followed by a scarlet fever-like eruption. In August he noted some black spots the size of a pinpoint on his body. The

pigmentation has gradually increased. From May 1933 to the present time the patient has received seven injections of neoarsphenamine, average dose 0.5 Gm., and twenty-four injections of 2 grains (0.12 Gm.) each of bismuth salicylate in oil.

He presents a generalized eruption consisting of round or oval, deeply pigmented, bluish-black macules varying in size from that of a pea to that of a bean. The eruption is most marked on the trunk.

A biopsy specimen is shown. The Wassermann reaction of the blood was 4 plus.

DISCUSSION OF TWO FOREGOING CASES

DR. E. C. GAGER, St. Paul: One probably will see more fixed eruptions from bismuth as time goes on because bismuth can certainly cause this type of eruption. Several weeks ago I happened to treat two men for plantar warts who were taking bismuth. I was surprised to see that after the administration of about one-half filtered skin unit (filtered through 3 mm. of aluminum) of roentgen rays these warts began to change in color, and inside of a week they were absolutely black. Bismuth has been recently advocated for the treatment of plantar warts, and this experience seems to me to indicate that bismuth may affect the papillary bodies and may become fixed in the skin.

DR. HAMILTON MONTGOMERY, Rochester: The histologic section in Dr. Sweitzer's case shows irregular clumps of bismuth deposited in the upper portions of the cutis without much appreciable inflammatory reaction. Even with hematoxylin and eosin stains this metallic deposit can be distinguished from melanin pigmentation which is also present.

The histologic section in the case of the Mexican with pigmentation resembles more that seen in fixed drug eruptions due to various medications. A deposit of heavy metal was not present in the skin sections, but there was a definite inflammatory reaction.

LYMPHOBLASTOMA (LEUKEMIA CUTIS). Presented by DR. JOHN F. MADDEN, St. Paul.

This man, aged 79, a jeweler, presents numerous bluish-red, firm, superficial, freely movable, painless nodules in the skin which vary in size from that of a pea to that of a silver dollar. This is accompanied by general lymphadenitis. The patient has lost 30 pounds (13.6 Kg.) in weight in the last six months. The spleen is not palpable. There has been no change in the blood picture.

A biopsy specimen is shown.

DISCUSSION

DR. E. C. GAGER, St. Paul: I treated this man six or seven years ago. He was referred to me by a surgeon who put a brace on his right leg; as a result of wearing this brace he had ordinary dermatitis and eczema of the leg which responded to the usual treatment and which he says has been well for five years. Now he has this eruption. I have had the same experience in two other cases. One, I remember particularly, was shown a year or so ago. That case also was diagnosed as leukemia; I treated the patient several years ago for staphylococcic infection resulting from a boil. I also recall a third case of this sort. I wonder whether some day one will not discover some serologic reaction in these cases that will give some indication of beginning lymphoblastoma or leukemia cutis. I wonder if any one has any idea as to the process which may be behind the development of the lymphatic disease.

DR. HAMILTON MONTGOMERY, Rochester: The histopathologic picture in this case is typical of lymphatic leukemia.

DR. JOHN BUTLER, Minneapolis: Were the lymphocytes large or small?

DR. JOHN F. MADDEN, St. Paul: They were small.

DR. FRANCIS W. LYNCH, St. Paul (by invitation): In connection with observations tending to show the presence of a leukemic background long before the

appearance of clinical leukemia, one may mention the occurrence of chronic lymphatic leukemia at about the same time in twin brothers (Dameshek, W.; Savitz, H. A., and Arbor, B.: Chronic Lymphatic Leukemia in Twin Brothers Aged Fifty-Six, *J. A. M. A.* **92**:1348 [April 20] 1929), which might indicate an influence extending as far back as the time of conception.

DR. HAMILTON MONTGOMERY, Rochester: In answer to Dr. Gager's question, any of the lymphoblastomas can occur primarily in the skin. Mycosis fungoides usually starts in the skin and sometimes spreads to the internal organs, whereas the other types of lymphoblastoma usually begin in the internal organs and spread to the skin. In view of the negative hemocytologic studies in this patient, it is possible that the disease started in the skin and will be apparent in the blood later. In a recent paper on exfoliative dermatitis and malignant erythroderma, I called attention to several cases in which apparently benign dermatoses preceded the development of lymphoblastoma. One patient had an exfoliative dermatitis, clinically apparently of a sensitization type because it promptly cleared up in the hospital. The histopathologic picture was strongly suggestive of lymphoblastoma. A year later the patient had a recurrence of the trouble, and when seen by Dr. Barney a short time after that he had typical mycosis fungoides both clinically and histologically.

LUPUS ERYTHEMATOSUS IN A COLORED PATIENT. Presented by DR. H. E. MICHELSON, Minneapolis.

This man, aged 49, states that two months ago circumscribed areas of hyperpigmentation appeared over the nose and cheeks, that they attained a certain size and then remained stationary, and that the color became much deeper in the surrounding skin. The lesions are disks of varying size, almost jet black, showing some dilated plaques but no atrophy. The microscopic section, however, showed the classic signs of lupus erythematosus.

DISCUSSION

DR. H. E. MICHELSON, Minneapolis: This is a most unusual case, and I think that the condition is superficial lupus erythematosus occurring in a colored man and that the most prominent change is a marked degree of hyperpigmentation. The microscopic observations substantiate the clinical diagnosis.

NEW YORK ACADEMY OF MEDICINE, SECTION OF DERMATOLOGY AND SYPHILIS

EUGENE TRAUB, M.D., *Secretary*

Feb. 5, 1934

LEO SPIEGEL, M.D., *Chairman*

MILIARY, PUSTULOSQUAMOUS DERMO-EPIDERMITE IN PROGRESSING CENTRIFUGAL PLAQUES (GOUGEROT'S DISEASE). Presented by DR. BOLESLAW LAPOWSKI.

This boy, aged 15, states that his present condition started in babyhood. He has been treated in various institutions, but no satisfactory record could be obtained from him.

The disease involves the scalp. He had complete alopecia, but the growth of the hair has returned, and he now has a good crop of hair with only here and there small crusted patches.

On the back of the neck, ears, forehead, eyelids, eyebrows, face, nares and edges of both lips are tiny follicular impetiginous lesions. On the bend of the

left elbow, bend of the left wrist, palms, extensor surfaces of the elbows, groins, genitals (except the upper portion of the penis, anus and buttocks), front and back of each knee, front of each leg, back of one foot and toes, can be seen impetiginous patches with shiny, distinct borders. The eruption is especially pronounced in the region of the genitals and penis, where the erythema is more vivid and desquamative. On the mucous membrane of the mouth and tongue are patches and lines looking like aphthae.

A brother of the patient has the same disease and all of the lesions on the patient's body correspond to a minor degree with those of his brother.

The urine was found to be free from sugar and albumin.

DISCUSSION

DR. J. GARDNER HOPKINS: I was struck by the similarity of Dr. Lapowski's case to one which I described in a paper on moniliasis and moniliids (ARCH. DERMAT. & SYPH. **25**:599 [April] 1932). The outstanding thing in my case was that one could reproduce in the boy's skin lesions similar to those which occurred spontaneously by the injection of a sterile extract of Monilia albicans. He carried this fungus in his intestines, and that seemed to explain the pathogenesis of the lesions. Dr. Lapowski is right in saying that it may not explain the whole syndrome. I am extremely interested in Dr. Lapowski's statement that Gougerot described the same clinical picture in a considerable number of cases. I am sorry that I have not seen Gougerot's reports.

DR. BOLESLAW LAPOWSKI: This patient is a brother of L. R., who was presented at the last meeting as showing Gougerot's disease. Since Dr. Hopkins' patient and mine are presented at this meeting, it can be asserted that the condition of Dr. Hopkins' patient is not the same as that of L. R., as erroneously reported at the previous meeting.

The patient (M. R.) presented tonight has suffered from lesions identical with those of his brother, L. R., which were described in the transactions of the January 2 meeting of the society (ARCH. DERMAT. & SYPH. **30**:144 [July] 1934). The condition started in his babyhood, and he has never been free from outbreaks. The patient and his brother were treated by Dr. W. Sachs from January 1932 to January 1933. No diagnosis was arrived at until Dr. Hopkins, seeing the two patients in the fall of 1932, reported the presence of Monilia in the skin and intestines of L. R., but only in the skin of M. R. and not in the intestines, as the boy failed to come in to be examined for Monilia in the intestines. L. R. was presented at the Manhattan Dermatologic Society on Jan. 10, 1933 (ARCH. DERMAT. & SYPH. **28**:124 [July] 1933), by Dr. Satenstein with a diagnosis of moniliasis.

To Dr. Hopkins must be given the credit of finding and reporting for the first time in cases of Gougerot's disease the presence of Monilia in the intestines and in the skin, as in the article by Staehelin (*Arch. f. Dermat. u. Syph.* **165**:313, 1932) full credit is not given to Dr. Hopkins.

DR. FRED WISE: Dr. Lapowski's discussion was both entertaining and instructive. I believe, however, that I am not making a mistake in saying that I do not think that this disease was first described in 1922 by Gougerot. Dermatologists had seen cases similar to these long before that date but did not know how to classify them as Gougerot did.

DR. MARION B. SULZBERGER: I hesitate to say anything after Dr. Lapowski's discussion, but I have to protect my friends against some of Dr. Lapowski's statements. The paper that Dr. Lapowski referred to is one by Staehelin, Yui Wu Mu and van Schouwen (*Arch. f. Dermat. u. Syph.* **165**:294, 1932), and the work was done in Switzerland while I was at the Zurich clinic. Dr. Hopkins was kind enough to send a copy of his paper to Dr. Bloch before its publication, and we added the references to Dr. Hopkins' work in the galley proofs of the Staehelin paper. The work was done and the paper completed before Dr. Hopkins' paper was received.

With regard to the question of the advances made in the study of this disease, Dr. Lapowski said that the important work was getting twenty-eight cases of this disease and studying them from the clinical point of view. I do not want to say anything against clinical descriptive dermatology, which is of course necessary and fundamental, but I think that Dr. Hopkins' work with Monilia may be more important than Gougerot's descriptions, giving a new approach to the pathogenesis, etiology and therapy of this condition.

DR. BOLESLAW LAPOWSKI: In order to establish the pathogenicity of Monilia in dermatologic cases, all the methods of examination have been used—first, bacteriologic cultures, then biologic tests, agglutination tests, complement-fixation tests and even allergic tests—but no method gave full satisfaction. At present, one tries to inject the monilia antigens into the skin in order to produce the same lesions which Monilia causes, but here the description varies, as the time of appearance of the lesions after the injection is important in ascribing the origin of the lesions to Monilia. Even the question of whether the organism in the intestines has the same biologic character as that in the skin is still open.

PSORIATIC ERYTHRODERMA WITH ARTHRITIC CHANGES. Presented by DR. LOUIS CHARGIN.

S. S., a man aged 27, American born, single, a refractionist, states that about three years ago he had an eruption on the elbows and knees. The eruption was dry and scaly and was declared by a physician to be psoriasis. Two and a half years ago a generalized eruption developed which has persisted. Whether this eruption had a relationship to therapy it is impossible to establish. There is now universal erythroderma with moderate scaling. When the erythroderma appeared the patient had arthritic pains, which first appeared in the ankles and later affected all the large and many of the smaller joints. The involvement of the joints lasted about seven months and was severe enough to keep the patient in bed most of the time. For three months there was an accompanying rise of temperature to from 101 to 103 F. In time there developed changes in the bone, as evidenced by the roentgenologic findings, which showed marked destruction as well as hypertrophic changes. The clinical findings, the course and the roentgenologic observations do not permit me to state that there was a definite interplay between the psoriasis and the arthritis, i. e., that the condition was psoriasis arthropathica.

Histologic examination of a section of skin showed psoriasis. Examination of the blood showed 5,200,000 red cells, and 16,350 white cells, with 79 per cent polymorphonuclears, 16 per cent lymphocytes, 4 per cent mononuclears and 1 per cent eosinophils; the hemoglobin was 90 per cent.

DISCUSSION

DR. HERMAN SHARLIT: I have always had the impression that generalized erythroderma complicating psoriasis was in some way causally bound up with therapy used in the treatment of the psoriasis. It is interesting to note that this patient did not receive treatment for the psoriasis and still this distressing transition set in.

DR. LOUIS CHARGIN: Dr. Sharlit is correct in his statement. Most patients with this disease give a history either of the application of local therapy or of the ingestion of some drug, but there are exceptions, and this patient's case is one.

A CASE FOR DIAGNOSIS (LYMPHOGRANULOMA? SARCOID?). Presented by DR. LOUIS CHARGIN.

A. S., a man aged 45, born in Italy, a watchman, states that his present trouble began one year ago on the left temple, where there is now a single large plaque, measuring from 2 to 3 cm. in diameter. Scattered about the scalp in the vicinity there are a number of small nodules, apparently part of the same process.

The Wassermann, Kahn and Kline tests were all negative. The blood count showed 91 per cent hemoglobin, 4,900,000 red cells and 10,200 white cells, with 50 per cent polymorphonuclears, 40 per cent lymphocytes, 9 per cent monocytes and 1 per cent eosinophils. The red cells were normal in size, shape and color.

Microscopic examination of a section of skin showed an infiltrating growth that spread between the connective tissue fibers throughout the entire depth of the section and was separated from the overlying epidermis by narrow zones of uninvaded corium. The connective tissue fibers were fragmented. The greater number of cells were uniform in size and shape. Between the cellular exudate many small pieces of collagen fibers took a basophilic stain. There was a vacuolated effect in parts of the section. This might have been due to the fixation. The majority of the cells were fibroblasts.

The Weigert stain showed absence of elastic tissue fibers, and the van Gieson stain showed that connective tissue fibers persisted and the exudate took a yellow stain. The carbol fuchsin stain failed to reveal tubercle bacilli. (The description of this slide fitted the other two sections.)

DISCUSSION

DR. FRED WISE: One would expect to make a diagnosis in this case from the histologic observations but I was disappointed in reading the histologic report. I could not make much out of it. On clinical grounds, I should not hesitate to make a diagnosis of lymphosarcoma.

A CASE FOR DIAGNOSIS (VESICULAR PSORIASIS?). Presented by DR. ISIDORE ROSEN.

E. R., a woman aged 39, gives an irrelevant past and family history. Her eruption began about thirteen years ago on the face and has run a varied course with remissions and recurrences.

The eruption is almost limited to the right side of the body, being present on the face, neck, shoulder region and forearm, but occurs also in the first left interdigital space. The clinical aspect varies. On the face and neck the lesions are vesicular, with a tendency to crust formation. On the chest and forearm they assume the form of lichenified plaques. During the course of observation there appeared a dollar-sized scaly patch on the pubic region, strongly resembling psoriasis. This persisted for three weeks and disappeared spontaneously.

On examination the urine was normal. The Wassermann reaction of the blood was negative. Examination of the blood revealed 92 per cent hemoglobin, 5,440,000 red cells and 16,700 white cells, with 54 per cent polymorphonuclears, 33 per cent mononuclears and 9 per cent eosinophils. The basal metabolic rate was + 19 per cent. Cystoscopic and pyelographic examination revealed no abnormalities of the genito-urinary tract. Several biopsy specimens from the chest, back and left thumb showed dermatitis with lichenification.

DISCUSSION

DR. HERMAN GOLDENBERG: Through the courtesy of Dr. Rosen I have been able to observe this patient since last September. Dr. Rosen and I have discussed the case frequently and at length, but have not come to any agreement as to diagnosis. I could never be convinced of the diagnosis of psoriasis, but labeled the condition neurodermatitis or eczema with lichenification.

DR. FRED WISE: There is no one in this room with whom I would rather agree on a clinical diagnosis than Dr. Rosen, but I cannot in this case. Whether the eruption is chronic eczema, neurodermatitis or a disease of exogenous origin, I do not pretend to say, but I can see no evidence of psoriasis.

DR. SIGMUND POLLITZER: When I was in Unna's Clinic in 1889-1890, my associates and I occasionally observed a case of a condition which we called "eczema seborrhoicum psoriasiforme." These were cases which showed a psoriatic

scaling on lesions with a seborrheic localization. I do not regard the present case as meriting this appelation.

DR. BOLESLAW LAPOWSKI: I congratulate Dr. Rosen. Vesicular psoriasis is a good clinical diagnosis. Gougerot in the last *Archives dermosyphiligraphique de la clinique de l'Hôpital Saint-Louis* published reports of two or three cases which he presented to Jadassohn; and Jadassohn answered by letter and gave his reasons why he called such eruptions psoriasis. Usually they are called eczema. Gougerot's description is of circinate, vesicular and pustular psoriasis. To my mind the case presented by Dr. Rosen corresponds to the cases of Gougerot. The histologic reports given by Jadassohn and Gougerot correspond to the picture of psoriatic lesions. I consider this eruption Jadassohn's type of psoriasis.

DR. ISIDORE ROSEN: I am glad to have presented this patient's case because of the many discussions that were elicited. I can readily understand that it would be impossible to establish a diagnosis by clinical examination of such an unusual condition. Dr. Goldenberg and I have been studying this patient for five months at various times, and we still differ as to the diagnosis. My diagnosis of vesicular psoriasis is based on the following points: the erythematous and firm character of the involved area, the micaceous scaling and the occasional deep-seated papulovesicles which rupture and are covered with an adherent crust. Further, the persistence of the lesions, the resistance to all forms of therapy and the presence of clinically characteristic psoriatic lesions on the hand and pubic region make me believe that the condition is an unusual form of this disease.

A CASE FOR DIAGNOSIS (SARCOID? LEPROSY?). Presented by DR. FRANK C. COMBES.

This man, aged 54, a sailor, for three years has had a well defined erythematous patch with an irregular border on the left side of his neck, extending onto the cheek bone. The lesion does not disappear on pressure and is irregularly anesthetic. The ulnar nerves are thickened. He has a coin-sized, slightly infiltrated lesion on the left upper arm and the left shoulder blade and atrophy of the left thenar eminence. There are no other manifestations suggesting leprosy.

The patient has spent many years at sea in tropical waters and has always enjoyed good health except for typhopneumonia one year ago. Lately his left ear has felt colder than the right.

Smears from three sections of tissue were negative for Bacillus leprae on repeated examinations. The Kahn reaction was 4 plus.

The process is superficial for the most part. Some sections on microscopic examination showed involvement of the deep cutis. The areas of infiltration consisted mostly of epithelioid cells, with some round cells and giant cells. The reticulum was loose and did not appear foamy.

DISCUSSION

DR. FRED WISE: I tested the lesions with a pin and found that the patient has distinct anesthesia, which is much in favor of a diagnosis of leprosy rather than sarcoid. I think that he has leprosy.

DR. FRANK C. COMBES: I hoped that the members might come to a definite conclusion in this case. The patient is in the hospital at Ellis Island, and the officials are anxious for a definite diagnosis. My first impression was that the condition was leprosy. The biopsy has caused all the confusion. In the early stages lepra is difficult to diagnose. Its presence can be verified only by the finding of the bacillus. Repeated examination of smears from three specimens, at which time twenty slides were studied, gave negative results. Repeated examinations of the nasal secretion were also negative. Possibly the only way to settle the diagnosis will be to send the patient to Carville where Dr. Denney can observe him over a prolonged period. The histologic picture is not conclusive for leprosy

or for any one type of sarcoid. Some sections look like the Darier-Roussy type while others in places suggest Boeck's type. The picture does not suggest typical leprosy. It resembles that of a tuberculid.

DR. T. J. RIORDAN: Three biopsies were performed. The first specimen showed the topography and cellular reaction of the Darier-Roussy type of sarcoid. The reticulum and some of the cells appeared foamy, a point in favor of leprosy. However, no lepra bodies were present, and examination of the tissue smears for the bacillus gave negative results. The second specimen showed the topographic features of the Darier-Roussy type of sarcoid; the third showed the superficial type, with giant, epithelioid and round cells. The last two did not have the foamy cells or the characteristic so-called lepra bodies. Smears were negative.

DR. SIGMUND POLLITZER: It seems to me that the condition does not look like the Darier-Roussy type of sarcoid, which is a sarcoid of the hypoderm with deep infiltration and a bluish-red color. This is not what is seen in this case. The patient has patches that might well be those of leprosy. He has an enlarged ulnar nerve and anesthesia. My strong impression is that the condition is leprosy. How were the sections fixed?

DR. T. J. RIORDAN: The first was fixed in a dilute solution of formaldehyde and the other two in alcohol. The specimen from the lesion on the face was removed without a local anesthetic. I thought that clinically the condition was leprosy, and I was disappointed in not being able to substantiate that diagnosis histologically. About thirty slides were examined for the bacillus.

DR. MARION B. SULZBERGER: There is a sarcoid type of leprosy which is a rare but definite form of the disease. I believe it was first described by Leloir in 1880. The histologic structure is like that of tuberculous sarcoid, and as in the latter condition, the bacilli are absent or extremely few, attenuated and difficult to find. Prolonged search is necessary in order to demonstrate them. I believe that this is such a case. J. Jadassohn has frequently described and spoken of tuberculoid leprosy. A description of this form will be found in V. Klingmüller's article in Jadassohn's "Handbuch der Haut- und Geschlechtskrankheiten" (Berlin, Julius Springer, 1930, vol. 10, pt. 2, p. 337).

DR. T. J. RIORDAN: In the cases of sarcoid leprosy are not the lepra cells and foam cells found?

DR. MARION B. SULZBERGER: The sarcoid form of leprosy is morphologically like the sarcoid forms of tuberculosis, both clinically and histologically.

DR. FRANK C. COMBES: Clinically I think that the condition is leprosy. There are two possible explanations of the difficulty in finding the organism: first, that mentioned by Dr. Sulzberger, that this may be a case of tuberculoid leprosy of Jadassohn; second, that it may be a neuroleprid, in which it is always difficult to find any organism.

DR. HERMANN FEIT: It is interesting to see the Darier-Roussy sarcoid type of leprosy. Dr. Wade of Manila, who has studied leprosy in all parts of the world, verified that a case was an unusual type of leprosy giving the impression of Boeck's sarcoid. The finding of the lepra bacillus seems to be difficult in both types. An allergy test with vaccine made from the lepra bacillus might be helpful in making a diagnosis.

DR. MARION B. SULZBERGER: This form of test is difficult in leprosy as one cannot get cultures of the bacilli. However, it has been carried out with extracts of lepromas, with positive results, corresponding to those obtained with tuberculin in tuberculosis.

BILATERAL MYXEDEMA OF THE LOWER PARTS OF THE LEGS FOLLOWING OPERATION FOR EXOPHTHALMIC GOITER. Presented by DR. LEWIS B. ROBINSON.

M. C., a woman aged 34, was first seen in 1930 by Dr. Sloan for symptoms of hyperthyroidism. Her basal metabolic rate was at that time + 32 and later + 51. A subtotal bilateral thyroidectomy was done in September 1930. Histologic sections

of the lobe showed a "marked hyperplastic tendency of the parenchyma cells." A diagnosis of exophthalmic goiter was made.

In May 1931 the basal metabolic rate was +9. There were no symptoms of thyroid dysfunction. In 1932 the Wassermann reaction was found to be 4 plus. A history was obtained that the patient had had a positive Wassermann reaction in 1925 and had received a few injections. She was given four injections of neoarsphenamine and three of a bismuth compound. The Wassermann reaction was negative and the patient was lost sight of until January 1934. At that time she gave a history of a swelling of the left leg for six months and of the right leg for a shorter time. There were no symptoms of hyperthyroidism. The Wassermann reaction was negative.

The entire dorsum of the left foot and the lower third of the left leg are about twice the normal size, and the skin is markedly thickened and verrucous, with accentuation of the follicular openings. At the proximal end of the swelling there are a number of discrete and coalesced, semitranslucent, firm tumors, noninflammatory. On pressure, the area is firm. There is no pitting edema or other symptoms. The right leg is similarly involved but to a lesser extent. For the past five days the patient has kept the feet elevated, and there has been a slight improvement.

At biopsy the cutis showed perivascular infiltration, and the collagen had entirely undergone myxomatous degeneration. The histologic diagnosis was "possibly myxedema."

DISCUSSION

DR. FRANK C. COMBES: Has Dr. Robinson any explanation for the edema? What is the functional disturbance in the thyroid gland which produces it?

DR. HERMANN FEIT: I saw a similar case presented by Dr. Galewsky in Copenhagen. This case proves that all such peculiar patches that one finds on the extremities should be examined carefully and different kinds of stains made, i. e., for mucin, amyloid and other metabolites. I think that the connection which Dr. Robinson makes between the operation for exophthalmic goiter and the hypothyroidism is good.

DR. FRED WISE: In the past four or five years there have been recorded in the literature a number of examples of circumscribed and diffuse myxedema of the skin associated with hyperthyroidism. I had the impression that the lesions were similar to these in distribution and localization, but that the epidermis was practically unchanged except as a result of pressure from edema and that the skin was rather smooth. This case is unique to me as far as the rough, dry and elephantiasis-like changes in the epidermis are concerned.

DR. EUGENE TRAUB: A case closely resembling the one presented tonight was shown at the recent Atlantic Dermatological Conference in Washington, D. C. That case also followed an operation for exophthalmic goiter, and the cutaneous changes were exactly similar.

DR. J. GARDNER HOPKINS: I think that hyperkeratosis, which may be almost verrucous, has been noted in many of these cases. I observed a patient in private practice who showed changes of this type and who also presented plaques of xanthoma on top of the myxedema plaques.

DR. HERMAN GOODMAN: Did the patient have the lesions on the legs before the thyroid gland was removed?

DR. HERMAN SHARLIT: Has it been the experience of the other members that patients with long-standing elephantiasis of the legs show the hyperkeratosis and pigmentation evidenced by this patient?

PIGMENTATION DUE TO ARSENIC AND LEAD? Presented by DR. EUGENE TRAUB.

H. G., a woman aged 43, Jewish, gives an irrelevant family history except that a maternal aunt was diabetic. The past history is unimportant except that she has had eczema on the legs at intervals since the age of 21.

Her present trouble began about July 1933 on the fingers and arms and then involved the scalp, chest, neck and face. The eruption was apparently severe, with much oozing, necessitating the application of wet dressings. She received, in addition, several exposures to roentgen radiation and various other types of local applications. At the time she was first seen she still presented a slight erythematovesicular eczema or dermatitis, most pronounced on the face, neck and arms.

A general examination revealed no foci of infection in the teeth, although one decaying tooth was present. Examination of the nose and throat gave negative results except that there was a suspicion of infection of the tonsils. Gynecological examination revealed nothing abnormal except slight retroversion of the uterus.

The Wassermann and Kahn reactions of the blood were negative. The basal metabolic rate showed an average of + 4.6 per cent. The blood pressure was 106 systolic and 80 diastolic, 105 systolic and 75 diastolic, and 110 systolic and 90 diastolic, on successive occasions. On routine examination, the urine was normal except for the presence of metals. The report was as follows: percentage of solids, 3.06; arsenic, 0.048 mg. per hundred grams, and lead, 0.049 mg. per thousand cubic centimeters.

The chemical examination of the blood showed: chlorides, 499.1 mg. per hundred cubic centimeters; sugar, 106 mg.; urea, 10.7 mg., and uric acid, 1.95 mg., and carbon dioxide, 59.8 cc. per hundred cubic centimeters of plasma. The blood count showed 67 per cent hemoglobin, 3,400,000 red cells and 6,800 white cells, with 56 per cent polymorphonuclears, 37 per cent lymphocytes, 3 per cent mononuclears and 4 per cent eosinophils. The sedimentation was within normal limits. Examination of the blood showed 0.777 mg. of arsenic and 0.29 mg. of lead per hundred cubic centimeters. Examination of the hair showed 0.16 mg. of arsenic per hundred grams and no lead. There was not sufficient tissue to make an examination for lead. There was 1.22 mg. of arsenic per hundred grams.

Biopsy showed an atrophic epidermis. The blood vessels in the upper corium, especially the capillary bodies, were dilated but not engorged. Slight edema was present. Below the upper third of the corium the tissue appeared normal. Pigment was scattered through the corium. The methylene blue stain showed the upper portion of the papillary bodies to contain numerous chromatophores associated with yellow-brown pigment. There was no pigment in the basal cell layer.

HYPERPIGMENTATION AND DEPIGMENTATION FOLLOWING RECURRENT ERYTHEMA MULTIFORME-LIKE ERUPTIONS. Presented by DR. SAMUEL M. PECK and DR. HENRY SILVER.

R. S., a colored woman aged 40, mother of three children, was admitted to the clinic on Jan. 11, 1934. For eleven months she had had "little swellings" on and under the surface of the skin of the chest, arms and body down to the hips. On the cheeks and lips there was diffuse erythema which did not include the chin and nasal area. The chest and arms presented diffuse pigmentation. The anterior surfaces of the arms showed irregular areas of pigmentation and erythema which tended to become confluent. The borders were elevated and sharply demarcated. On the chest and arms were areas of depigmentation (leukoderma). On the arms posteriorly were many isolated, pale, violaceous-red lesions which were edematous and sharply circumscribed.

On January 20, the erythematous lesions had become brownish (a sort of mahogany brown); scaling was taking place in some, while others appeared to be fading and becoming flat.

Since a history of drug ingestion had been obtained, the patient was given 1 grain (0.06 Gm.) of phenolphthalein, with negative results.

Biopsy on one of the acutely inflamed lesions showed hyperkeratosis but no parakeratosis. The lower layers of the epidermis showed both intercellular and intracellular edema with invasion of leukocytes at various points. The underlying cutis was edematous and contained many dilated blood vessels showing perivascular infiltration of leukocytes and lymphocytes. Pigment was seen in clumps

free in the tissues as well as in chromatophores. The histologic picture suggested toxic erythema or erythema multiforme.

The blood count on January 25 showed 4,500,000 red cells and 10,900 white blood cells, with 62 per cent polymorphonuclears, 35 per cent lymphocytes and 3 per cent mononuclears.

As the erythematous lesions have faded, a number have become hyperpigmented. No areas of depigmentation have been seen to develop. The patient was given phenobarbital, ¼ grain (0.0015 Gm.) three times a day, with no exacerbation of the lesions. From the histologic picture it is evident that owing to the inflammatory reaction in the epidermis a sort of "incontinentia pigmenti" is taking place, which accounts for the leukoderma.

A Case for Diagnosis (Pigmentation Due to Arsenic?). Presented by Dr. E. Kelley.

M. A., a colored girl aged 19, born in Panama, has had a generalized eruption for six months. In July 1933 she noticed raised and itching lesions on the left arm. These remained unchanged for from five to six weeks, and then similar lesions appeared all over the body. In time the lesions disappeared, leaving dark red spots which did not cause itching. No new lesions have appeared in the past four months.

There are now blackish patches, not raised above the surface of the skin, which in some places show a distinctly serpiginous outline and in other locations, particularly over the entire back, form large confluent areas. The mucous membranes are free.

Examination of the urine and skin for the presence of arsenic (on account of one provocative injection of arsphenamine after the eruption had developed) was done. The report was as follows: urine, 0.03 mg. of arsenic in a twenty-four hour specimen (total volume, 670 cc.); blood, 0.14 mg. per hundred grams of dry specimen, and skin, 0.10 mg. per hundred grams of dry specimen.

DISCUSSION ON THREE FOREGOING CASES

Dr. E. William Abramowitz: An eczematous eruption caused by arsenic is apt to be followed by hyperpigmentation of the skin. The spotted pigmentation occurring following the use of inorganic arsenic and not preceded by any previous eruption is usually of the "raindrop" variety and is not apt to be as dark as that noted in some of the cases presented tonight. The finding of arsenic in the blood and urine and also in the skin, even in amounts above the normal, must be taken in conjunction with other evidences of arsenical hypersensitiveness or intoxication. For instance, it is most important in such cases to demonstrate that the patient is hypersensitive to arsenic.

Dr. Louis Tulipan: In reference to the case in which lead and arsenic were found, I think that one should exclude lead as the etiologic agent. Lead *per se* does not produce cutaneous changes, e. g., pigmentation. When it is the cause of a toxic condition, one finds definite signs, such as the blue-black line in the gums, stippling of the red blood cells, colic and nervous symptoms.

Dr. Hermann Feit: The finding of lead does not prove that it has anything to do with the pigmentation disturbance. Hyperpigmentation and depigmentation are found frequently in colored people without any definite disorder.

Dr. Abraham Walzer: I think that the pharmacologic action of lead and the immunologic action of this metal have been confused. A patient who has sensitivity to lead does not necessarily give signs or symptoms of an excessive intake of lead. The toxic-pharmacologic symptoms will occur in every patient after a certain quantity has been ingested, but the immunologic symptoms will occur only in those sensitized to this metal and do not necessarily imply a quantity. The smallest amount might cause allergic symptoms.

DR. PAUL GROSS: I am interested in Dr. Traub's patient because the condition does not seem to me a simple pigmentation, especially on the neck, where one can see a lichenoid dermatitis, and the whole picture suggests the diagnosis of melanodermatitis toxica. Probably this has resulted from a prolonged use of ointments. A biopsy on tissue from the neck might clear the diagnosis.

DR. EUGENE TRAUB: One section was removed from the neck and stained in the ordinary way, while another section was reserved for a determination of arsenic. Unfortunately, enough tissue was not obtained to permit an examination for lead also. Something over one hundred and fifty times the normal arsenic content was found in the skin, and about five times the normal amount of lead was found in the blood. This, together with the finding of both arsenic and lead in the urine and hair, strongly suggests that the retention of these metals may play a causative rôle in the hyperpigmentation. The pigmentation is a recent complication of several months' duration, but the history of a recurring dermatitis of some kind on the neck and arms has covered a period of about three years. Several of the members of this section have treated my patient for these attacks, giving her various ointments and several treatments with the roentgen rays. Although the pigmentation followed these recent roentgen exposures, I do not believe that the irradiation had anything to do with the hyperpigmentation. I shall be glad to submit a slide to Dr. Gross.

FOX-FORDYCE DISEASE. Presented by DR. SAMUEL M. PECK.

This girl, aged 19, born in the United States, eighteen months ago began to have a series of deep abscesses in both axillae. These were drained and this was soon followed by itching in the axillae and the development of the present condition. A great deal of roentgen therapy had been applied to the axillae but had had no effect on the course of the eruption.

In both axillae there are fairly well defined areas which consist of skin-colored to bluish-red lichenoid papules, follicular and nonfollicular in distribution. The affected areas feel distinctly edematous and there are several well defined abscesses at the periphery. There are similar papular lesions in the areola of the right breast and around the anus. The vaginal mucosa and mons are free, although there is a great deal of itching. The chief complaint is the intense pruritus with a tendency to secondary infection and abscess formation. The scars noted in the axillary region are due to attempts at therapy by electrodesiccation of individual papules.

MANHATTAN DERMATOLOGIC SOCIETY

MIHRAN B. PAROUNAGIAN, M.D., *Secretary*

Feb. 13, 1934

HOWARD FOX, M.D., *Chairman*

DERMATITIS FACTITIA. Presented by DR. HOWARD FOX.

This woman, aged 23, first noticed an eruption four and a half months ago, after having worked for ten days in an aluminum factory. This was the first time she had been employed, and according to her statement the conditions at the factory were satisfactory and agreeable. Her duty consisted in trimming the edges of aluminum cans. While she was at work four splinters of aluminum entered the soft tissue of the front of the left wrist. One of the splinters was removed by the patient on the following day, and the others were extracted by her physician. Two days later the entire wrist became swollen, tender and

red, and at the end of a week an ulcer the size of a dime appeared. This gradually enlarged to the size of a quarter.

When I saw her on Dec. 4, 1933, there was a deep oval ulcer about 1½ inches (3.5 cm.) long, showing an elevated tender border and a highly inflammatory appearance. There was considerable redness as well as swelling of the entire front of the wrist, interfering with the motion of the wrist joint. The ulceration had been curetted and treated by ultraviolet irradiation and various ointments.

General examination showed a mentality below normal, absence of pharyngeal and corneal reflexes and some anesthesia to pin prick on the hands and forearms. From these stigmas of hysteria, from the artificial appearance of the eruption and from the fact that it was on the left wrist (the patient being right-handed), a diagnosis of factitious dermatitis was made. Her family physician was advised to protect the lesion from interference on the part of the patient by a plaster cast. This was done, five casts being applied at weekly intervals. When the patient was seen on Feb. 6, 1934, there were two new, highly inflammatory small ulcers. It was then learned that the casts had not been applied so that all outside interference with the lesions was prevented.

DISCUSSION

DR. GEORGE M. MACKEE: I agree with Dr. Fox that the patient is a malingerer. Such patients are exceedingly resourceful, and it is often necessary to go to extremes in order to establish a diagnosis. I suggest that the patient be hospitalized and that a plaster of paris cast be applied to the entire hand and the entire forearm. There should be no window in the cast. In addition, the patient should be kept under constant observation both day and night. She should not be allowed even to go to the toilet alone. She must be observed every minute. The reason for such advice is that I have had several patients in whose cases the correct diagnosis was established only after this method was employed.

DR. PAUL E. BECHET: There is no doubt that occasional cases of this type of eruption have a sexual connection.

DR. GEORGE M. LEWIS: How long should such a cast be left on?

DR. GEORGE M. MACKEE: It may be necessary to keep the parts in a cast for several weeks. I usually remove the cast after the first few days and immediately reapply it. It is again removed in perhaps four or five days and reapplied. This procedure is continued for two or three weeks.

DR. LUDWIG OULMANN: In a case of this kind, with formation of granulations, I think it advisable to apply Unna's gelatin bandage first and apply the plaster cast over it.

DR. MIHRAN B. PAROUNAGIAN: Is this a compensation case? In such cases the patients often practice this kind of malingering.

A CASE FOR DIAGNOSIS (LYMPHOBLASTOMA?). Presented by DR. GEORGE C. ANDREWS.

O. W., a white man aged 34, has had lumps on the scalp several times during the last three years. Some of these disappeared spontaneously and others after the taking of arsenic given by Dr. Oppenheim in Vienna. The previous attacks resembled the present one, except that the lesions were not so large.

In the left frontal region there is a group of indurated, erythematous tumors, 1 cm. or more in diameter, of one month's duration. The lesions are firm to palpation and do not fluctuate; they are not tender. On the vertex there is a similar group of smaller lesions, and on the left side of the back of the neck there is a subcutaneous firm mass that is probably a swollen lymph gland. The Wassermann reaction was negative. A blood count, biopsy and roentgenogram of the skull were made today, but the reports have not been received.

DISCUSSION

DR. J. FRANK FRASER: I believe that the condition should be classified as belonging to the malignant lymphoid hyperplasias. It is difficult to say which type without a microscopic examination. It may prove to be mycosis fungoides of the d'emblée type.

DR. DAVID L. SATENSTEIN: The fact that the lesions responded to arsenic should be taken into consideration. From the character of the lesions I offer a tentative diagnosis of some form of sarcoid, possibly the Spiegler-Fendt type. I do not favor a diagnosis of mycosis fungoides.

DR. ISADORE ROSEN: I suggest that in addition to the diagnoses offered some form of sarcoid be considered; that diagnosis can be made only by further histologic examination.

DR. GEORGE M. MACKEE: It is, of course, impossible to make a definite diagnosis on the basis of the clinical features. I agree with Dr. Fraser that the condition may well belong in the lymphoblastoma group. It may be mycosis fungoides d'emblée. Occasionally this disease begins with nodules or tumors which may remain in one region for months or years. The fact that in this case the lesions have disappeared and reappeared several times in the same region over a period of a few months certainly seems to rule out any type of neoplasm. Naturally, syphilis must be definitely excluded.

DR. HOWARD FOX: As the results of the laboratory tests are not yet available, it is impossible to make a positive diagnosis. The clinical appearance, in my opinion, is suggestive of one of the lymphoblastomas. The condition may be mycosis fungoides, leukemia, lymphosarcoma or Hodgkin's disease. The lesions look similar to those in my case of Hodgkin's disease of the scalp, in which the diagnosis was confirmed histologically, but they may prove to be any one of the other diseases mentioned.

DR. GEORGE C. ANDREWS: I first thought that the condition was tertiary syphilis, but as the Wassermann test was negative the possibility of lymphoblastoma or sarcoid seems tenable.

BOWEN'S PRECANCEROUS DERMATOSIS. Presented by DR. DAVID L. SATENSTEIN for DR. GEORGE M. LEWIS.

Mr. A. W. aged 28, a chauffeur, was first seen on Dec. 22, 1933, when he complained of a lesion on the glans penis of five months' duration. There were no subjective symptoms. The lesion when first noticed was approximately the same size and depth as now.

There is a history of the occasional ingestion of phenolphthalein. He has never taken arsenic therapeutically. There are no lesions on other parts of the body.

There is a roughly triangular lesion (the sides measuring approximately 1, 1.5 and 1.5 cm.) on the left side of the glans penis. The area appears elevated, but this impression cannot be confirmed by palpation, as the lesion is as soft as the surrounding skin. The color is reddish to violaceous and is readily removed by diascopic pressure.

Histologic study disclosed marked hyperplasia of the epidermis, the cells making up the greater part of the process being dyskeratotic. The cells were markedly irregular in size. The nuclei were of various sizes, and some multinucleated cells were present. The majority of the nuclei were hyperchromatic. A considerable number of mitotic figures at various stages were noted. The granular layer was still present. The basal cell layer as an entity was not present.

DISCUSSION

DR. GEORGE M. MACKEE: As the clinical features are insufficiently characteristic for an unqualified diagnosis, it is necessary to depend on the micro-

scopic observations. I examined one slide a few days ago. There was an abnormal proliferation of basal cells within the epidermis. In fact, there was a proliferation of both basal and squamous cells, the former predominating. This, together with some dyskeratosis, led to a diagnosis of intra-epidermal epithelioma. As I saw no clumping of cells or nuclei, I was unwilling to make a diagnosis of Bowen's carcinoma. However, at the present moment I cannot say that that diagnosis is incorrect. I prefer to beg the question and call the condition an intra-epidermal carcinoma.

DR. J. FRANK FRASER: The chief histologic features of Bowen's disease, clumping of the nuclei (epithelial giant cells) and hydropic mitoses, were not present in the section that I examined. Bowen's disease is an intra-epidermal carcinoma of the prickle cell type with an intact basal layer. There has been no convincing proof offered that this lesion is a carcinoma.

DR. MAX SCHEER: Clinically it is impossible to make a diagnosis in this case. One has to rely on the histologic picture, uncertain as it is.

DR. DAVID L. SATENSTEIN: If the condition had been accepted clinically and treated as lichen planus, the histologic observation of a picture other than that of lichen planus would be considered wrong. There are many instances in which the clinical features are those of lichen planus but the microscopic observations do not corroborate that diagnosis. This case raises the question as to whether the clinical concept of lichen planus is to be checked by the microscope or the microscopic observations are to be ignored when they do not agree with the clinical diagnosis.

Must one accept the diagnosis of lichen planus even if in the microscopic examination one finds evidence of, for example, an intra-epidermal epithelioma or epithelioid tubercles with or without an inflammatory reaction, a definite perivascular plasma cell infiltration, etc.?

DR. JOSEPH J. ELLER: I agree with Dr. MacKee and Dr. Fraser that the condition is an intra-epidermal carcinoma.

LYMPHOBLASTOMA. Presented by DR. ISADORE ROSEN.

R. M., a white woman aged 30, married, born in Poland, first attended the skin clinic of the New York Post-Graduate Medical School and Hospital on Jan. 29, 1934. She presents an ill defined umbilicated papular eruption with surrounding zones of erythema on the arms, axillae, front of the chest and thighs. In the left supraclavicular area there are two olive-sized subcutaneous nodules. The patient gives a history of having had glands in these sites excised one year ago.

Examination of the blood showed: red cells, 4,000,000; hemoglobin, 86 per cent; white cells, 6,500; polymorphonuclears, 64 per cent; lymphocytes, 35 per cent, and mononuclears, 1 per cent. The general morphologic characteristics of the blood cells were normal.

DISCUSSION

DR. DAVID L. SATENSTEIN: The histologic changes in the cutaneous lesions were those of a degenerative process in the vessel walls. There was nothing to corroborate the clinical diagnosis of lymphoblastoma. I believe that the lesions on the arms are toxic.

DR. GEORGE M. MACKEE: I agree with the diagnosis of lymphoblastoma. Clinically the condition looks like either Hodgkin's disease or lymphatic leukemia. The eruption on the extremities presumably is toxic and may be placed in the category of a leukemid.

DR. J. FRANK FRASER: The section that Dr. Satenstein examined was from the skin. The cutaneous lesions accompanying lymphoblastoma in the majority of cases are fundamentally different histologically from the lesions in the lymph nodes. To establish a diagnosis in this case it will be necessary to examine sections from a lymph node.

DR. LOUIS B. MOUNT: One must remember that there are two types of eruptions in this group. One type is purely toxic, consisting of a variety of lesions which show no characteristic pathologic changes. The other type, a real lymphoblastoma of the skin, shows the characteristic pathologic picture of the disease.

GENERALIZED LUPUS VULGARIS IN A SYPHILITIC PATIENT. Presented by DR. HOWARD FOX.

Mrs. A. R., colored, aged 28, has been married for eleven years, during which time she has had three miscarriages and no living children. About one year ago her husband had a penile lesion which healed quickly. The Wassermann reaction of his blood was negative. The patient applied for advice at the Harlem Hospital, where her blood was found to give a strongly positive Wassermann reaction. There were no clinical manifestations at that time. She was then given antisyphilitic treatment, and after the third injection of neoarsphenamine an eruption appeared on the face and neck and soon involved in addition the trunk and extremities. New lesions continued to appear during a course of forty-eight injections of neoarsphenamine and a bismuth preparation.

The eruption is generalized, somewhat sparse and roughly symmetrical. There are no subjective symptoms. All of the lesions are dull red, and none of them are ulcerated. They are present in the region between the nose and eyes, on the nostrils, vermilion borders of the lips and nape of the neck and scattered over the trunk and extremities. Some of them are elevated, rounded nodules the size of a pea; others are the size of a pinhead (on the nape of the neck especially). Some of them are flattened elevations suggesting at first glance lupus erythematosus. Others have the appearance of small keloids. Those on the face are rather typical of lupus miliaris disseminatus faciei. Histologic examination of a lesion from the back showed the structure of tuberculosis. The case will be reported later in detail.

DISCUSSION

DR. DAVID L. SATENSTEIN: I believe that this is the first case that I have seen in which lupus vulgaris developed during active antisyphilitic therapy. It is suprising that these lesions developed under therapy which is supposed to benefit tuberculous lesions.

DR. ISADORE ROSEN: I suggest that a biopsy specimen be taken from one of the active lesions on the face and stained for tubercle bacilli and that part of the tissue be injected into a guinea-pig. It is sometimes difficult to differentiate between syphilis and tuberculosis histologically, and further corroboration by animal inoculation is needed to substantiate the diagnosis.

DR. JOSEPH J. ELLER: This is one of the most interesting cases that I have seen for a long time. One would expect an active focus of infection somewhere. In spite of the fact that the lesions have not improved under antisyphilitic treatment. I suggest that further tests be made to rule out syphilis. I have observed cases in which syphilitic lesions developed while the patient was under active antisyphilitic treatment. This might be one of those resistant cases.

DR. GEORGE C. ANDREWS: Although my impression is that the patient has lupus erythematosus disseminatus, I believe that it is important to demonstrate the tubercle bacillus, as that is the only way to differentiate the condition positively from syphilis. Therefore, it is important to inoculate a guinea-pig and search the sections for the tubercle bacillus.

DR. FRED WISE: I agree with the diagnosis as presented.

DR. GEORGE M. MACKEE: I believe that the condition is cutaneous tuberculosis, probably lupus vulgaris with an unusual amount of connective tissue overgrowth. I believe, moreover, that the eruption is entirely independent of syphilis, which the patient has in addition.

DR. MAX SCHEER: I agree with Dr. MacKee. The presence of syphilis does not exclude the possibility of tuberculosis.

DR. HOWARD FOX: I agree that the only two means of proving the condition to be tuberculosis are demonstration of the bacilli in the lesions and the obtaining of positive evidence by guinea-pig inoculation. The latter procedure is certainly more convenient, as it is well known that in searching for the tubercle bacillus an average of fifty or sixty slides must be examined to find one organism.

TROPHIC ULCER. Presented by DR. MAX SCHEER.

P. D., a man aged 55, Russian, an unemployed tailor, was referred from the surgical department to the dermatologic department of the clinic of the New York Post-Graduate Medical School and Hospital with a history of having been operated on for tic douloureux in December 1933. Section of the sensory roots was done, and anesthesia of the right side of the face resulted.

Three weeks ago the present condition developed. The right ala of the nose is reddened and moderately swollen and presents a sharply punched-out, indurated ulcer involving the free edge of the alar cartilage. The ulcer is anesthetic, as is the rest of the right side of the face. The patient also presents a single well defined groove on the distal third of each finger-nail, and he says that this condition has been present only since he was operated on.

DISCUSSION

DR. LOUIS B. MOUNT: I agree with the diagnosis as presented.

DR. GEORGE M. MACKEE: Basically the condition probably is a trophic ulcer. However, there is considerable induration; this may be the result of infection with pyogenic organisms, or carcinoma may be developing. I think that the case deserves further study. The changes in the nails are bilateral, unusual and interesting. I do not know the cause of this anomaly.

LUPUS ERYTHEMATOSUS (TELANGIECTATIC TYPE)? DERMATITIS MEDICAMENTOSA (GOLD)? Presented by DR. MAX SCHEER.

J. L., a white man aged 32, from the skin clinic of the New York Post-Graduate Medical School and Hospital presents a roughly round, serpiginous patch about 4 by 2 inches (10 by 5 cm.) on the right cheek, extending to the left side of the nose. The edges are red and scaly, but the center is clear except for some atrophy and several dilated, plugged follicles. The patient has received at least fifty injections of gold sodium thiosulphate and fifteen of a bismuth preparation. For the past two weeks he has taken the Hollander treatment with some improvement.

Four weeks ago, after the dose of the gold preparation was increased from 75 to 100 mg., several red, dry, scaly, round and serpiginous patches appeared on the legs. Some of the earlier lesions have faded, but new ones are appearing. Spectroscopic examination of a section from the left leg showed a large amount of gold. Histologic examination of a patch from the left leg showed dermatitis but no evidence of lupus erythematosus.

DISCUSSION

DR. LUDWIG OULMANN: The patient has had relapses whenever the gold preparation was injected. Dry, scaly patches are present. To my mind, there can be no doubt that the condition is a dermatitis due to gold.

DR. MIHRAN B. PAROUNAGIAN: I favor the diagnosis of gold dermatitis.

DR. J. FRANK FRASER: I favor the diagnosis of gold dermatitis and suggest that the injections of gold sodium thiosulphate be discontinued.

DR. GEORGE M. LEWIS: I treated this patient several months ago, and at that time there was no sign of eruption on the legs.

DR. MAX SCHEER: I was informed that the patient had a similar eruption some time ago. The administration of the gold preparation was discontinued.

When the injections were stopped, the condition cleared up, only to reappear when they were renewed. There is no reason why there may not be an atypical location in this disease as well as in many others.

LEUKOPLAKIA. Presented by DR. MIHRAN B. PAROUNAGIAN.

CASE 1.—A. Z., a man, aged 45, presented himself first on Jan. 31, 1934, complaining of an asymptomatic lesion at the left angle of the mouth of six months' duration. He had a penile sore twenty-one years ago, for which he received two injections into the arm and also used mercury inunctions for three or four years. Since then he has had no treatment.

At the angle of the mouth there is a semicircular lesion with a raised, dark red border, a slightly depressed and ulcerated center and some yellowish crusting. This lesion extends slightly onto the mucous membrane. There is a small ulcer on the inner aspect of the lower lip, near the angle. On the buccal mucosa bilaterally, on the inner aspect of the lower lip and on the front of the tongue, there are discrete patches of leukoplakia.

The Wassermann reaction on Jan. 31 was 4 plus. He has been given one injection of mercury and one of neoarsphenamine.

CASE 2.—M. B., a man, aged 50, was admitted to the Bellevue Hospital clinic on Jan. 22, 1934, complaining of soreness of the tongue and mouth of two months' duration.

He denies having had venereal disease. He has smoked about ten cigarets daily for the past thirty years.

There are diffuse patches of leukoplakia on the front half of the tongue and also some patches on the buccal mucosa and the upper and lower lips. In the center of the tongue, anteriorly, there is a flat, slightly elevated, firm lesion, which is nonulcerated. On the upper lip, developing from a patch of leukoplakia, is a small papillomatous, nonulcerated excrescence. His Wassermann reaction on January 22 was 4 plus. He has received two injections of a bismuth preparation and two of neoarsphenamine and shows slight improvement.

CASE 3.—C. F., a man aged 49, first came to the Bellevue Hospital, outpatient department, on Dec. 30, 1931, with a history of having received in the previous two and a half years one hundred intravenous and ninety intramuscular injections. He had no history of chancre; he had had one attack of urethritis twenty-five years before. He received treatment because of an accidental finding of a 4 plus Wassermann reaction.

At the time of admission the only lesion found was extensive leukoplakia of the tongue and buccal mucosa. The Wassermann reaction of the blood was found to be 4 plus, and he was given a course of injections of mercury and neoarsphenamine, followed by a course of injections of thiobismol. After this the Wassermann reaction was 2 plus. He then received a course of injections of bismuth and neoarsphenamine, and his Wassermann reaction became plus-minus, as it has remained to the present time.

On the distal half of the tongue are diffuse patches of leukoplakia. There are similar but smaller patches at the angles of the mouth, extending about 1 inch onto the buccal mucosa.

DISCUSSION

DR. JOSEPH J. ELLER: Leukoplakia may become aggravated and serious. If it disappears with discontinuance of smoking, it is best to leave the patient alone. Desiccation is the safest form of treatment if treatment is indicated.

DR. GEORGE M. MACKEE: These three patients have more than leukoplakia. In each case there is a combination of leukoplakia, smooth atrophy and interstitial glossitis. Any one or two of these features have no special etiologic significance, but the triad is considered by most observers to be fairly pathognomonic of syphilis. The second patient has a dime-sized vegetating area in the center of the tongue which should be excised or radically destroyed by electrosurgery because it may be a beginning carcinoma.

In each case the leukoplakia involves the mucous membranes of the cheeks and the tongue. It would be impracticable to remove all of the leukoplakia with radium or by surgical methods, and it is unnecessary to do so. Such patients should be examined every three months. Any time that danger signals occur—ulcers, fissures, verrucous or vegetating lesions, etc.—such an area should be destroyed. Occasionally, when a wide area shows dangerous signs, a plastic operation is indicated.

DR. LOUIS B. MOUNT: The patients should be observed at definite intervals and only when something appears which looks extremely suspicious should anything radical be done. Oral hygiene is important. Syphilis, if present, should be treated.

DR. DAVID L. SATENSTEIN: I believe that leukoplakia develops independent of smoking, of poor oral hygiene and of syphilis. There must be some underlying conditions, as yet undiscovered, which favor the development of these lesions. There is nothing in the pathologic process which indicates the underlying etiologic factor.

DR. ISADORE ROSEN: I do not believe that all cases of leukoplakia of the tongue and buccal mucosa are due to syphilis. I had under observation a lawyer, aged 24, with extensive interstitial glossitis and leukoplakia of the tongue and inner surfaces of the cheeks of a number of years' standing. His personal history and family history revealed no evidence of syphilis, and all clinical and serologic investigations gave negative results. On examination I found that he had arsenical pigmentation of the trunk and arsenical keratosis of the palms and soles which were due to the ingestion of arsenic in all forms administered over a period of years for a chronic pruriginous skin disease which he had in early life.

Regarding the treatment of Dr. Parounagian's patients, I am of the opinion that they should not receive any of the arsenicals intravenously; the condition is best controlled with the mildest form of antisyphilitic therapy.

DR. HOWARD FOX: The presenter stated informally that he was showing these patients to prove the relationship of the condition to syphilis. There can be no doubt that in a considerable proportion of cases leukoplakia is preceded by syphilis. Statistics in this regard vary greatly, from 30 per cent as reported by Max Joseph to 80 per cent as cited by Fournier. In my series of forty cases the incidence was 65 per cent, which corresponds roughly to the figures of numerous combined statistics. While syphilis is only a contributing cause, in almost every case of buccal leukoplakia there is some long continued local irritation, usually tobacco smoke. The disease is infinitely more common in men than in women, though with the present amount of smoking done by women there may be a different story to tell in the future. As to treatment, I feel that there is often a meddlesome attempt to do the impossible. In mild beginning cases, all that is needed is to advise the patient to exercise proper oral hygiene, to stop smoking and to remain under medical observation. In cases involving the greater part of the mouth, radical attempts to remove the entire process are out of the question. When, however, ulceration, fissures or nodules appear, it is time for immediate vigorous action by destructive methods. I am opposed to the unnecessary production of scars in the mucous membrane of the mouth, as these may become the starting point of cancer.

DR. MIHRAN B. PAROUNAGIAN: When there are a positive Wassermann reaction and a history of syphilis, I believe in giving antisyphilitic treatment. I believe in treating the patients as one does those with any other form of syphilis. I also instruct the patients as thoroughly as possible about oral hygiene and give them mild mouth washes. I could show a dozen patients whom I have treated in whom from 50 to 70 per cent improvement was noticed. I do not think that there is any occasion to be alarmed about treatment with arsenicals in these cases.

NEW ENGLAND DERMATOLOGICAL SOCIETY

J. HARPER BLAISDELL, M.D., *Secretary*

Feb. 14, 1934

RUDOLPH JACOBY, M.D., *President*

A CASE FOR DIAGNOSIS. Presented by DR. JOHN G. DOWNING, Boston.

This white boy, aged 2 years, has had birth marks on both axillae since birth, according to the mother's statement. At five months, a small nodule was noted on one breast under the skin, but not adherent. The nodule increased in size during the next three weeks, after which the skin presented bluish mottling. It then regressed. Since that time the patient has had other nodules on the anterior portion of the chest, axillae and inner aspects of the upper parts of the arms. One broke down and was evacuated. The nodules have never been tender.

On each axilla there is a vascular nevus which occupies an area about 2½ inches (6 cm.) square. On the anterior portion of the chest, axillae and inner aspects of the upper parts of the arms are subcutaneous tumors which vary in size from that of a small grape to that of a walnut. They are soft, nontender and irreducible by pressure.

The parents have refused to permit excision of one of the nodules. A complete examination of the blood revealed nothing abnormal.

The patient has received no treatment.

The diagnosis suggested is vascular nevi with possible formation of hemangio-endothelioblastoma.

DISCUSSION

DR. JOHN G. DOWNING, Boston: Many physicians speak about the disappearance of tumors. I saw a girl three weeks ago who had a rather remarkable history. She had had one breast removed at the hospital for tumor formation several years before. The diagnosis made at that time was hemangiosarcoma. Lesions subsequently developed in various parts of the body, and when I saw her the other breast was a solid mass of tumors, and scattered over all the body were various soft, fluctuating growths, the overlying skin presenting definite blueness. These hemangio-endothelioblastomas in their earliest stages apparently come and go. They fill up, disappear and finally remain when they have sufficient tissue in their walls. The girl was in perfect health, not disturbed by the presence of the tumors, and would have no further treatment. She had had excision and roentgen and radium therapy, with resultant radiodermatitis and disfiguring scars.

DR. WILLIAM P. BOARDMAN, Boston: It is news to me that such lesions come and go without apparent cause.

DR. J. H. SWARTZ, Boston: This is a far-fetched guess, but I suggest the possibility that the boy presented has calcinosis. I suggest a study of the blood calcium and excision of one of the lesions for biopsy.

LICHEN PLANUS ATROPHICUS. Presented by DR. ARTHUR M. GREENWOOD, Boston.

L. B., a colored woman aged 49, has had an itchy eruption for six months. The general examination gave negative results.

There is an extensive eruption on the body and the proximal parts of the extremities; it is papular and shows a tendency to streaking. It has a definite atrophic appearance with small, cornified, blackish, comedo-like plugs in the center of many lesions. Examination of the mouth revealed nothing abnormal.

Biopsy showed lichen planus atrophicus.

DISCUSSION

DR. ARTHUR M. GREENWOOD, Boston: I think that those atrophic lesions frequently clear up. That is surprising when one sees the section.

DR. FRANCIS M. THURMON, Boston: Although the histologic picture corresponds to that of lichen planus atrophicus, clinically the lesions with the black comedo-like centers also correspond to keratosis follicularis contagiosa.

DR. E. MYLES STANDISH, Hartford, Conn.: Follicular plugs in atrophic lichen planus are common.

DR. CLARA P. FITZGERALD, Worcester, Mass.: Do the lesions appear to be more unusual because the patient is colored?

DR. FRANCIS M. THURMON, Boston: My associates and I have had two cases of this kind in the clinic at the Boston Dispensary. The patients were younger than this woman, both being girls between the ages of 8 and 10 years. Those were the first cases that I had seen. I judged them purely by the textbook descriptions of Andrews and Sutton. They responded clinically to the eruption caused by ointment containing red mercuric iodide.

DR. J. H. SWARTZ, Boston: Was atrophy present in the cases that Dr. Thurmon just cited?

DR. FRANCIS M. THURMON, Boston: As the lesions tended to clear there were white, shiny atrophic lesions for a time, but these eventually cleared and were replaced by normal skin. The atrophy was not permanent.

A CASE FOR DIAGNOSIS (TRICHOTILLOMANIA?). Presented by DR. E. LAWRENCE OLIVER, Boston.

This white boy, aged 8 years, four months ago noticed that his hair was falling out. The condition spread laterally. He had scarlet fever three years ago. General examination gave negative results. There is thinning of the hair, mainly to the left of the midline. There are no exclamation point hairs. There is a bald spot 3 by 5 cm. in diameter.

Microscopic examination for tinea gave negative results.

The patient has been given sulphur tablets, 10 grains (0.6 Gm.), three times a day. Improvement was noted at first, but the area is now spreading.

The patient is presented for diagnosis.

DISCUSSION

DR. J. HARPER BLAISDELL, Boston: I should accept the diagnosis of trichotillomania with considerable reluctance. The mother states that she first noted the lesions on the front of the forehead, where the hair is now the longest; one morning, while combing the child's hair, she found an area about 2 inches (5 cm.) round that was shiny and completely bald. If that is a fact, it would rule out this diagnosis. In the regions where the hair is now the shortest, numerous broken hairs can be seen. The mother has never discovered the child pulling out the hair. She has never found any hair on the pillow or on the bed in the morning.

DR. J. H. SWARTZ, Boston: I think the mother gave Dr. Blaisdell a story different from the one that she gave me. She is much against the diagnosis. She told me a few months ago that she found bunches of hair in the bed. This child and two others in the family have a neurologic background. The patient is bright in school, more so than the average child, and is active.

DR. MILDRED RYAN, Brockton, Mass.: The patient had no typical alopecia areata when I saw him. He bites his finger-nails. He is of the neurotic type. He gave a history of pulling out his hair.

DR. E. LAWRENCE OLIVER, Boston: I think that there is one other objection to the diagnosis I made tentatively; i. e., the involved area is so sharply defined

at the borders that it is difficult to imagine the child pulling the hair out over such a clearcut patch. When I heard of the family history that two brothers were psychopathic, I thought that was in favor of this diagnosis. Frequently such patients eat the hairs. They are apt to be very secretive.

DR. ELLWOOD C. WEISE, Bridgeport, Conn.: A roentgen examination of the stomach might show presence of a ball of hair.

DR. E. MYLES STANDISH, Hartford, Conn.: Dr. Oliver spoke about the fact that the lesions of trichotillomania are not apt to be sharply limited. I had a man who knew that he was pulling out the hair over a typical wedge-shaped patch on the forehead. The borders were so straight that they looked as though a ruler had marked them off.

MULTIPLE BENIGN SARCOID OF THE SKIN. Presented by DR. JOHN G. DOWNING, Boston.

This white woman, aged 37, was first seen on May 16, 1933. She stated that in August 1932 she was frightened by a dog, which caused a rather severe nervous reaction, and that six weeks later a red patch covered with pimples appeared on her left cheek. Later, these pimples appeared all over her face and on her arms and legs. All day at her work as a secretary she sat in the sunlight which came through a glass window; the eruption seemed to affect the areas which were exposed, for on the legs it stopped at the hem of her dress and at the top of her shoes. There was no itching, burning or painful sensation.

Physical examination showed a well developed and well nourished woman. The lesions were practically confined to exposed areas, appearing on the face, a V-shaped band on the chest, the back down to the angle of the scapulae, the anterior surfaces of the lower thirds of the lower parts of the legs and the dorsa of the feet. The area covered by a wrist watch was free from eruption. The entire surface of the face was markedly erythematous and was studded with reddish-brown, flat papules. The eruption on the arms, chest, neck and legs was symmetrical and consisted of discrete papules varying in diameter from 3 to 5 mm. On the forearms and lower parts of the legs they were more or less flattened, but on the upper parts of the arms, chest and neck they were larger and more elevated. They were depressed in the center and were softer on palpation than those on the forearms, legs and face. The surface was smooth. There was no sign of any scaling. On pressing these lesions with a glass, there was a tendency toward an apple jelly color. The lesions on the lower parts of the arms and lower parts of the legs were reddish brown, but those on the upper parts of the arms and the chest were bright yellow. The patient stated that she had always been well but lately had noticed marked shortness of breath. At times she felt as if she were choking. An internist found slight enlargement of the thyroid.

The Wassermann reaction of the blood was negative. The blood count showed 4,680,000 red cells, 8,950 white cells and 82 per cent hemoglobin. The blood smear was normal. The urine was normal. The blood pressure was 138 systolic and 84 diastolic. The blood sugar was 121 mg. per hundred cubic centimeters. The basal metabolic rate was +18, +21 and +24 per cent on different occasions.

Biopsy was performed, and section of the skin showed histologically numerous small lesions, lying for the most part in the upper portion of the corium only a slight distance below the epidermis. Often these were found to have formed around sweat glands. They were somewhat oval in cross-section, and the larger ones were about 1 mm. in length. Under high power magnification these were found to be made up of fairly large cells with achromatic nuclei and acidophilically staining cytoplasm, probably monocytes. In many places these had fused to form multinucleated giant cells similar to those found in tuberculous lesions.

Roentgen examination of the chest showed that the bony thorax was symmetrical and normal. Both domes of the diaphragm were normal, and the heart and the great blood vessels were normal. Both lungs were filled with a large

amount of coarse, irregular mottling with a good deal of fibrosis. This process extended to the tops of the lungs on both sides. It was probably extensive fibroid tuberculosis involving the greater part of both lungs. Films of the hands showed no evidence of any definite pathologic changes in the bones.

The patient has been observed intermittently since her first visit. On Nov. 14, 1933, she stated that the shortness of breath had entirely disappeared. She felt more rested, and the rash had greatly improved. She had also gained 4 pounds (1.8 Kg.) in weight. The papules have now disappeared from the face and forearms. The face shows little sign of a papular eruption, but there is mild erythema. The papules have disappeared from the forearms and the lower parts of the legs, leaving definite pigmentation. However, the papular lesions on the upper parts of the arms, neck and chest are practically unchanged.

Treatment has consisted mostly of rest and small doses of iron, arsenic and strychnine.

DISCUSSION

DR. MYER M. TOLMAN, Chelsea, Mass.: My associates and I had a patient with multiple lesions of the chest and back. A diagnosis of sarcoid was made. A few lesions were desiccated here and there, and new ones reappeared. He came under the observation of a physician who took roentgenograms and found evidences of tuberculosis in the chest. All the lesions disappeared and the patient gained about 20 pounds (9 Kg.) in weight with rest in bed and the general treatment for tuberculosis.

SCLERODERMA (CIRCUMSCRIBED). Presented by DR. E. LAWRENCE OLIVER, Boston.

This woman, aged 45, first noticed a patch on her left thigh five months ago. It was essentially asymptomatic. A general medical check-up revealed no trace of a former mitral stenosis and vaginal cyst.

On the left thigh there is an area 5 by 8 cm., circumscribed and irregularly oval, in which there is thickening of the skin with a "porky" feeling, surrounded by a violaceous border 14 by 17 cm. There is a similar "porky" feeling area on the left ankle. Pitting edema is present.

The basal metabolic rate was —8 per cent.

The patient has been given solution of pituitary for ten days.

DISCUSSION

DR. E. LAWRENCE OLIVER, Boston: My associates and I have been using solution of pituitary in such cases of scleroderma, and the patients have done remarkably well. I observed a child who had a palm-sized lesion on the abdomen which disappeared when she was given rather large doses of solution of pituitary daily. If the preparation is given slowly there seems to be little danger of adverse reactions. Dr. Izar, an Italian physician, reported a cure fifteen years ago in a case of scleroderma in which the patient was treated by injections of pituitary extract.

DR. JOSEPH MUELLER, Worcester: I have seen some instances of striking success in the treatment of scleroderma of this type in Budapest by Dr. Sellei. He is using a mixture of extract of pancreas and other tissue extracts. I do not know whether his extract is available in this country. I think not.

DR. WILLIAM P. BOARDMAN, Boston: I think that extracts of almost all glands have been used in cases of scleroderma. Sometimes syphilis is at the bottom of the disease, indirectly affecting the ductless glands. So many cases have been reported in the literature in which ductless glands have been affected that one has had to consider them. Not only pituitary should be tried, but one gland extract after another.

A Case for Diagnosis (Keratosis Follicularis?). Presented by Dr. Henry Benjamin, Springfield, Mass.

This white man, aged 30, presents lesions which have appeared gradually, first on the back and then on the chest, over a period of fifteen years. They are slightly itchy when the patient perspires.

There are discrete, dry, firm, brownish, papular lesions from 1 to 2 cm. wide at the mouths of follicles, symmetrically distributed over the back and chest. In the clavicular regions there are faded, mottled, brownish lesions.

Scrapings and cultures were negative for fungi. Biopsy showed hyperkeratosis, many large, swollen dyskeratotic cells and proliferation of epithelium at the mouths of follicles.

The patient has received no treatment.

DISCUSSION

Dr. Vincent Ryan, Providence, R. I.: This case is of unusual interest to me because of the peculiar pigmentation. I cannot classify the eruption as Darier's disease. The distribution and the duration suggest that it was caused by some medication given during childhood. However, I was unable to elicit any such information. An area on the right shoulder has become lichenified as a result of scratching. I classify the condition as an abnormal pigmentation of the skin.

Dr. Harvey P. Towle, Boston: I am willing to accept the diagnosis of keratosis located in the follicles. I should like to know what to do in such a case.

Dr. J. H. Swartz, Boston: The microscopic picture is much in favor of Darier's disease. The section shows (1) hyperkeratosis, (2) invaginations in the epithelium filled with horny material, (3) acanthosis and finger-like projections of the right cones, (4) a few suggestive corps ronds and (5) inflammation about the hair follicle. The clinical picture is not characteristic because of the absence of lesions on the forehead, chest and other typical areas.

Dr. Joseph Mueller: A good many of the lesions do not seem to be in the hair follicles.

Dr. John G. Downing, Boston: I saw the patient, and I think that I suggested the idea of keratosis follicularis. The microscopic picture agrees with this diagnosis rather well. It corresponds more with the type of keratosis follicularis described by Morrow, a very mild type, than with the type described by Darier and White. I could find no typical round bodies histologically.

Dr. E. Lawrence Oliver, Boston: It seems to me that the microscopic picture is consistent with follicular keratosis of some sort, but I question the diagnosis of Darier's disease. I do not know how to classify the condition.

Book Reviews

The Modern Treatment of Syphilis. By Joseph Earle Moore. Price, $5. Pp. 535. Springfield, Ill.: Charles C. Thomas, Publisher, 1933.

This book is a description of current practices and results in the treatment of syphilis, with special reference to Moore's experience in the Johns Hopkins Clinic. His methods and conclusions are based on the study of 18,000 cases in the syphilis division of the medical clinic, where there was ample opportunity to check the clinical and laboratory methods. Although the works of others are not overlooked, the author's entire statement of the subject is based on his own experience with this material. His consideration of the treatment of syphilis is full and complete. No details of the subject fail to receive consideration, and neither a beginner nor an expert will seek in vain for information on any topic relating to the treatment of syphilis. Moore emphasizes the importance of thorough examination of the patient before beginning treatment and careful attention to the patient's general condition during treatment. His emphasis on the patient rather than on routine is fortunate, as is his attitude that the physician should always keep in mind that he is treating patients as well as trying to cure syphilis. This is notably illustrated in the chapters on cardiovascular syphilis, neurosyphilis and syphilis complicated by pregnancy which are among the best in the book. Moore looks at syphilis from the point of view of the internist—of the very competent and conservative internist, in his case—and his work has the advantages of that point of view. He thinks that syphilis, if it is made a specialty, should be a specialty of the internist, and he himself offers a good argument for that position, because he has had an enormous amount of experience with syphilis and is familiar with dermatoses. The reviewer follows him to a certain extent in that direction. He thinks, for example, that visceral syphilis, cardiovascular syphilis and syphilis of the nervous system belong primarily to the physicians whose special concern is these various fields. But he thinks that those who, like dermatologists, are more engaged in the treatment of syphilis in general than the internists are, have the unprejudiced opinion that, with internists in general, syphilis of internal structures is best treated by the internists in association with syphilologists who have had wide experience in treating syphilis.

Moore does not entirely escape this criticism himself. With leukoplakia buccalis, for example, he takes the position (and quotes Bloodgood, a surgeon, in support of it) that this condition is so frequently associated with syphilis that it is to be practically accepted as a manifestation of it. This is a familiar error. Leukoplakia is no more a definite entity than eczema is and is no more in itself an indication for syphilitic treatment than eczema is for treatment with arsenic. Any competent dermatologist will tell him that leukoplakia is common in the mouth in all sorts of conditions that cause patches of chronic inflammation; that anything causing a patch of chronic inflammation of the mouth, from lichen planus to a rough tooth, may produce patches of it; that it is simply a manifestation of chronic inflammation and parakeratosis.

But this is a great book and a real contribution to the knowledge of syphilis. It is that rare sort of book in which the intelligent conclusions of an author who speaks from an enormous amount of experience of his own are given freely. The fulness of its detailed consideration of all topics relating to syphilis makes it particularly valuable as a reference book in perplexing problems in this disease, but its reading in entirety is worthy of any physician's time.

Wilhelm Conrad Roentgen and the Early History of the Roentgen Rays. By Otto Glasser, with personal reminiscences by Margaret Boveri. Price, $6. Pp. 494. Springfield, Ill.: Charles C. Thomas, Publisher, 1933.

This biography of Roentgen will be of interest to dermatologists. The first part is taken up with topics that primarily concern Roentgen: the discovery of the rays, Roentgen's first communications, the sensation that the discovery produced and an account of Roentgen the scientist and the man, first by the author and then in the form of personal reminiscences by Margaret Boveri. These intimate considerations of Roentgen and his work take up approximately the first 200 pages. In the next 250 pages are considered almost every topic that has a bearing on the history of the roentgen rays themselves and their scientific development and application, such as fluoroscopy, the use of the rays in medical diagnosis and in warfare, their physiologic effects, the development of apparatus and similar topics and, finally, a bibliography. This section is a treasury of historical information on roentgenology. The author has done well. His style is smooth and interesting; the subjects have been systematically arranged and developed, and the book not only is informative but also makes attractive reading. The author is enamored of his subject, which is a good trait in any biographer, but this causes him occasionally to make an overstatement, such as "he [Roentgen] was being surrounded by a glory such as no man of science had experienced previously." Newton, Franklin, Jenner, Faraday, Pasteur and Lister had experienced glory just as dazzling.

The book is a fine addition to the literature of roentgenology and deserves both purchase and reading. The publisher has done his part well and has produced a handsome volume.

Traité de la syphilis. Volume V. Publié sous la direction de Ed. Jeanselme. Price, 200 francs. Pp. 760, with 106 illustrations and 4 colored plates. Paris: Gaston Doin & Cie, 1934.

This fifth volume on syphilis, published under the editorial direction of Jeanselme, continues to show the high quality of the four previous volumes. The contributors are among the best known French dermatologists. This volume is devoted to visceral syphilis and covers the subject with the meticulous attention to detail that is characteristic of French dermatology. The table of contents gives an idea of the completeness of the book: Beginning with syphilis of the trachea, it includes a discussion of syphilis of the respiratory tract, of the mediastinum, of the cardiovascular system, of the endocrine glands and of the pancreas, syphilis and diabetes, syphilis in disturbances of nutrition, syphilis of the ear, nose and accessory sinuses, syphilis of the larynx, syphilis of the visual apparatus, syphilis and traumatism, surgical syphilis and syphilis and associated dieases—especially syphilis and tuberculosis, syphilis and alcoholism and syphilis and cancer.

In considering previous volumes, reference to the high character of this work has repeatedly been made. The work is an encyclopedia of syphilis, and this volume lives up to the high standard set by the previous ones. It is a credit to the editor and his collaborators and to French dermatology.

La lèpre. By E. Jeanselme. Quarto volume. Price, 600 francs. Pp. 668. Paris: Gaston Doin & Cie, 1934.

Professor Jeanselme's extensive researches in Paris and French Indo-China have enabled him to write an authoritative and comprehensive review of leprosy. His consideration covers the history, geography, epidemiology, semeiology and treatment, as well as the effects of the changing traditions and concepts concerning this disease on the social and hygienic practices of the persons affected. He illuminates all the topics by the interpolation of illustrative histories of cases and analyses of his findings. Although he is a dermatologist, he emphasizes constantly the outstanding importance of the neurologic manifestations. His discussion of the

diagnosis is particularly valuable because of his extensive knowledge of syphilis and of other neurologic and dermatologic syndromes. After a thorough consideration of the treatment, he laconically summarizes the therapy thus: There is no specific remedy for leprosy, but it is possible to ameliorate the symptoms, to arrest its course and perhaps even to produce radical healing of the infection by a combination of therapeutic measures in which cooperation is indispensable. These are hygiene, the treatment of concomitant maladies, internal and local treatment of leprosy as such and the treatment of complications. He believes that infection is transmitted from man to man by contact, and he gives this postulate of the dominant rôle in his discussion of prophylaxis.

Books Received

LE TRAITEMENT DE LA SYPHILIS RÉNALE. A. Sezary. Price, 8 francs. Pp. 53. Paris: J. B. Baillière et fils, 1934.

LA HEMOCLASIA DIGESTIVA COMO ÍNDICE DE INSUFICIENCIA HEPÁTICA. Juan Antonio Montoya O. Pp. 136. Tesis, Universidad de Antioquia, 1933.

Directory of Dermatologic Societies *

FOREIGN

British Association of Dermatology and Syphilology (Canadian Branch)

W. R. Jaffrey, President, 64 Hughson St., S., Hamilton, Ont.
Basil Bowman, Secretary-Treasurer, 408 Medical Arts Bldg., Hamilton, Ont.
Place: Montreal, Quebec. Time: May 4, 1935.

Royal Society of Medicine, Section of Dermatology

Henry MacCormac, President, 23, Wimpole St., W. 1, London.
W. N. Goldsmith, Secretary, 6, Upper Wimpole St., W. 1, London.

NATIONAL DERMATOLOGIC SOCIETIES

American Medical Association, Scientific Assembly, Section on Dermatology and Syphilology

Jeffrey C. Michael, Chairman, 1215 Walker Ave., Houston, Texas.
Harry R. Foerster, Secretary, 208 E. Wisconsin Ave., Milwaukee.
Place: Atlantic City. Time: June 10-14, 1935.

American Dermatological Association

Charles M. Williams, President, 114 E. 54th St., New York.
W. H. Guy, Secretary, 500 Penn Ave., Pittsburgh.

SECTIONAL DERMATOLOGIC SOCIETIES

Baltimore-Washington Dermatological Society

Harry F. Anderson, President, 1610, 20th St. N.W., Washington, D. C.
Joseph V. Kennedy, Secretary, 1835 Eye St., Washington, D. C.

Central States Dermatological Association

Joseph Brumberg, President, 528 Delaware Ave., Buffalo.
William F. Hoover, Secretary, 333 Linwood Ave., Buffalo.
Place: Buffalo. Time: 1934.

Iowa and Western Illinois Dermatological Association

J. C. Kessler, President, University Hospital, Iowa City.
Robert E. Jameson, Secretary, 1014 First National Bank Bldg., Davenport, Iowa.

New England Dermatological Society

Rudolph Jacoby, President, 270 Commonwealth Ave., Boston.
J. Harper Blaisdell, Secretary, 5 Bay State Road, Boston.

Southern Medical Association, Section on Dermatology and Syphilology

Thomas W. Murrell, Chairman, 17 E. Grace St., Richmond, Va.
C. C. Dennie, Secretary, Professional Bldg., Kansas City, Mo.
Place: San Antonio, Texas. Time: November, 1934.

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* Secretaries of dermatologic societies are requested to furnish the information necessary for the editor to make this list complete and to keep it up to date.

STATE DERMATOLOGIC SOCIETIES

Florida Society of Dermatology and Syphilology

Chairman for each meeting is elected from city in which meeting is held.
Elmo D. French, Secretary, 602 Huntington Bldg., Miami.

Louisiana Dermatological Society

J. A. Devron, President, 150 Baronne St., New Orleans.
M. T. Van Studdiford, Secretary-Treasurer, 912 Pere Marquette Bldg., New Orleans.

Medical Society of the State of New York, Section on Dermatology and Syphilology

George M. Fisher, Chairman, 264 Genesee St., Utica.
Frank Combes, Secretary, 80 W. 40th St., New York.

Medical Society of the State of Pennsylvania Section on Dermatology

Herbert J. Smith, President, 3303 N. 17th St., Philadelphia.
Robert L. Gilman, Secretary, 1930 Chestnut St., Philadelphia.

Michigan State Medical Society, Section on Dermatology and Syphilology

Robert C. Jamieson, Chairman, 1551 Woodward Ave., Detroit.
A. R. Woodburne, Secretary, Grand Rapids Clinic, Grand Rapids.
Place: Battle Creek. Time: September, 1934.

Minnesota Dermatological Society

D. D. Turnacliff, President, 407 Medical Arts Bldg., Minneapolis.
L. H. Winer, Secretary, 522 Medical Arts Bldg., Minneapolis.
Time: First Wednesday in October, December, February and April.

Oklahoma Dermatological Association

W. A. Showman, President, 108 W. 6th St., Tulsa.
C. L. Brundage, Secretary, 505 Osler Bldg., Oklahoma City.

Texas Dermatological Society

C. F. Lehmann, President, 303 E. Houston St., San Antonio.
Everett R. Seale, Secretary, 1215 Walker Ave., Houston.

LOCAL DERMATOLOGIC SOCIETIES

Atlanta Dermatological Society

Francis G. Jones, President, 384 Peachtree St., Atlanta, Ga.
Charles A. Wilkins, Secretary, Medical Arts Bldg., Atlanta, Ga.

Bronx Dermatological Society

Louis Chargin, Chairman, 1 W. 85th St., New York.
Henry Silver, Secretary, 290 West End Ave., New York.

Brooklyn Dermatological Society

Arthur Persky, President, 1750 Ocean Parkway, Brooklyn.
George F. Price, Secretary, 884 Lincoln Place, Brooklyn.
Time: Third Monday of each month except June, July, August and September.

Buffalo-Rochester Dermatological Society

Earl D. Osborne, President, 471 Delaware Ave., Buffalo.
Richard L. Saunders, Secretary, 33 Linwood Ave., Buffalo.

CHICAGO DERMATOLOGICAL SOCIETY

James H. Mitchell, President, 25 E. Washington St., Chicago.
Max S. Wien, Secretary, 104 S. Michigan Ave., Chicago.

CINCINNATI DERMATOLOGICAL SOCIETY

Harry L. Claassen, President, Provident Bank Bldg., Cincinnati.
Raymond G. Senour, Secretary-Treasurer, 19 W. 7th St., Cincinnati.

CLEVELAND DERMATOLOGICAL SOCIETY

R. E. Barney, President, 1021 Prospect Ave., Cleveland.
E. W. Netherton, Secretary, 2020 E. 93d St., Cleveland.

DETROIT DERMATOLOGICAL SOCIETY

H. L. Keim, President, 10 Witherell St., Detroit.
William G. Saunders, Secretary-Treasurer, 9203 Grand River Ave., Detroit.

LOS ANGELES DERMATOLOGICAL SOCIETY

Moses Scholtz, Chairman, 1930 Wilshire Blvd., Los Angeles.
Nelson P. Anderson, Secretary, 2007 Wilshire Blvd., Los Angeles.

MANHATTAN DERMATOLOGIC SOCIETY

Louis B. Mount, Chairman, 256 State St., Albany, N. Y.
Mihran B. Parounagian, Secretary, 126 E. 39th St., New York.

MONTREAL DERMATOLOGICAL SOCIETY

Alberic Marin, President, 1414 Drummond St., Montreal, Canada.
Paul Poirier, Secretary, 2073 St. Denis St., Montreal, Canada.

NEW YORK ACADEMY OF MEDICINE, SECTION OF DERMATOLOGY AND SYPHILIS

Ray H. Rulison, Chairman, 145 E. 54th St., New York.
E. William Abramowitz, Secretary, 853, 7th Ave., New York.

NEW YORK DERMATOLOGICAL SOCIETY

Fred Wise, President, 200 W. 59th St., New York.
R. H. Rulison, Secretary, 145 E. 54th St., New York.

OMAHA DERMATOLOGICAL SOCIETY

J. W. Hellwig, Chairman, 1319 Farnam St., Omaha.
Donald J. Wilson, Secretary, 1216 Medical Arts Bldg., Omaha.

PHILADELPHIA DERMATOLOGICAL SOCIETY

Robert L. Gilman, Chairman, 1930 Chestnut St., Philadelphia.
Vaughn C. Garner, Secretary, Greene and Coulter Sts., Philadelphia.

PITTSBURGH DERMATOLOGICAL SOCIETY

Bernhard A. Goldmann, President, Jenkins's Arcade, Pittsburgh.
J. Cavanaugh, Secretary, 28 Market St., Youngstown, Ohio.

ST. LOUIS DERMATOLOGICAL SOCIETY

Richard S. Weiss, President, 3720 Washington Blvd., St. Louis.
M. F. Engman, Jr., Secretary-Treasurer, 3720 Washington Blvd., St. Louis.

SAN FRANCISCO DERMATOLOGICAL SOCIETY

H. J. Templeton, President, 3115 Webster St., Oakland, Calif.
John M. Graves, Secretary, 909 Hyde St., San Francisco.

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ARCHIVES OF PATHOLOGY—Monthly. A periodical devoted to the publication of original contributions in the field of pathology, with abstracts from foreign and domestic literature, book reviews, the transactions of special societies, etc. Illustrated. Annual subscription price (two volumes): Domestic, $6.00; Canadian, $6.60; foreign, $7.00. Single copies, 75 cents.

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